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THIRD SERIES FOUNDED BY EDWARD JACKSON

CONTENTS

Ocular wound healing ..... *John H. Dunnington and Ellen F. Regan* 167
Lens-induced uveitis and glaucoma ..... *S. Rodman Irvine and Alexander Ray Irvine, Jr.* 177
Priscoline: A new vasodilator ..... *John J. Stern* 187
Retinal response to priscoline ..... *M. R. Cholst, H. F. Schilback, M. B. Handelsman and L. M. Levitt* 191
Implantation cyst of conjunctiva ..... *E. W. Perkins* 196
Pseudoglioma ..... *T. E. Sanders* 199
Syndrome of glaucomato-cyclitis ..... *Edwin Billet* 214
Retroplacement of inferior oblique ..... *Conrad Berens, Helen Grady Cole, Souren Chamichian, and Marjorie V. Enos* 217
Observations on vitamin deficiencies ..... *Max Edward Pohlman and Edward Francis Ritter, Jr.* 228
Autodemonstration of nystagmus ..... *Gordon L. Walls* 231
Visual screening of school children ..... *Elton R. Yasuna and Leah Sanders Green* 235
Menière's disease ..... *Harry McGrath* 241
LeConte's book on "sight" ..... *Beulah Cushman* 245
Trichlorethylene as an analgesic ..... *Bertha Offenbach* 250
True polycoria ..... *Norman S. Jaffe and Paul Knie* 253
Moebius's syndrome ..... *J. R. Stansbury* 256
Paradoxal ocular motility ..... *James Miles O'Brien* 261

DEPARTMENTS

Society Proceedings. 263 Correspondence ... 272 Abstracts 281
Editorials ..... 268 Book Reviews ..... 276 News Items 298

For complete table of contents see advertising page xv

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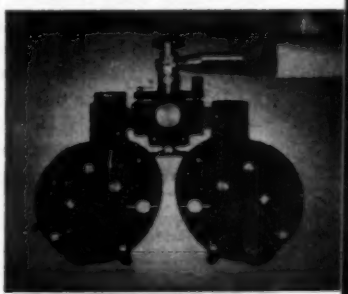

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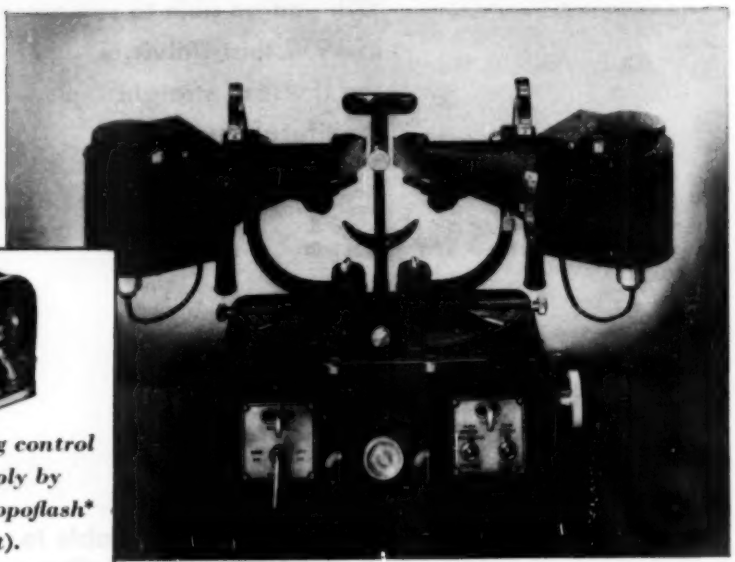

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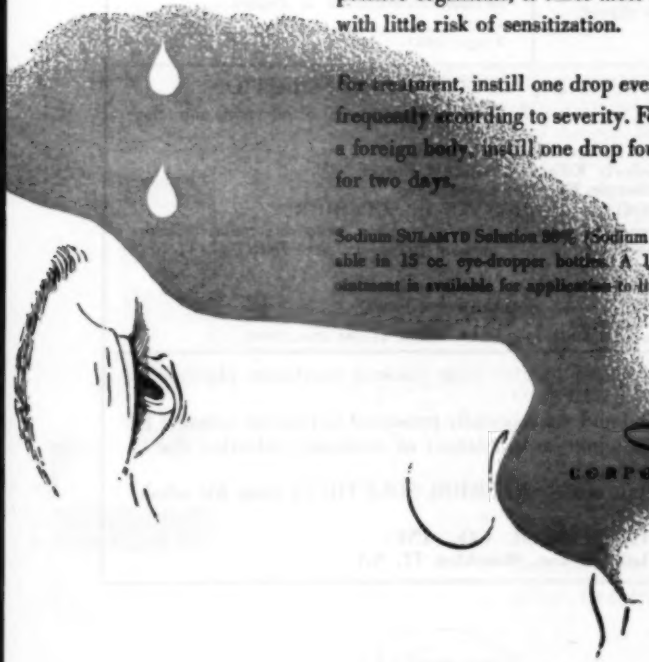

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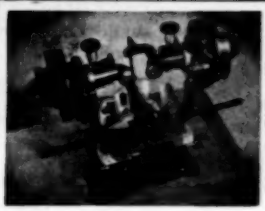

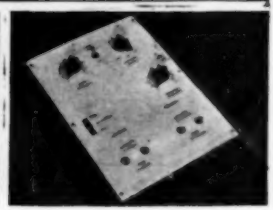

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Figure 73; Page 107

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Let us take a correction that reads: Right eye +1.00, left eye —1.00. This combination of corrections would produce a vertical imbalance of 1.6, because the left eye produces .8 base up and the right eye .8 base down, the resulting imbalance being 1.6.

Again, if the right eye required +1.00 and the left eye +2.00, there would be an imbalance of .8, due to the fact that at the visual point the right lens produces .8 base up and the left lens produces 1.6 base up, the difference being the amount of induced imbalance.

It will be seen that when one eye requires plus and the other eye requires minus, the total of the imbalance is found by adding the two prism values. When both are plus or minus, the imbalance is found by subtracting the lesser from the greater.

So far we have talked only about spherical corrections. If, and there generally is, a cylinder to be calculated, a special chart is required. This chart will be published in the April issue of the "Scientific Corner," followed by an explanation in the June issue. The "Scientific Corner" will appear every other month until this series is completed.

AMERICAN JOURNAL OF OPHTHALMOLOGY

SERIES 3 · VOLUME 35 · NUMBER 2 · FEBRUARY, 1952

CONTENTS

ORIGINAL ARTICLES

The effect of sutures and of thrombin upon ocular wound healing. The Jackson Memorial Lecture. John H. Dunnington and Ellen F. Regan 167
Lens-induced uveitis and glaucoma: Part I. Endophthalmitis phaco-anaphylactica. S. Rodman Irvine and Alexander Ray Irvine, Jr. 177
A new vasodilator in ophthalmology. John J. Stern 187
The response of the retinal vessels to priscoline in various vascular conditions. Study I. Mortimer R. Cholst, Harold F. Schilback, Milton B. Handelsman, and Leon M. Levitt 191
Implantation cyst of the conjunctiva. E. W. Perkins 196
Pseudoglioma. A clinicopathologic study of 15 cases: Part II. Pathologic classification. T. E. Sanders 199
Syndrome of glaucomato-cyclitic crises: Report of a case. Edwin Billet 214
Retroplacement of the inferior oblique at its scleral insertion. Conrad Berens, Helen Grady Cole, Souren Chamichian, and Marjorie V. Enos 217
Observations on vitamin deficiencies: In an Eye, Ear, Nose, and Throat Clinic of a Japanese prison hospital. Max Edward Pohlman and Edward Francis Ritter, Jr. 228
An autodemonstration of the "physiologic nystagmus." Gordon L. Walls 231
An evaluation of the Massachusetts vision test for visual screening of school children. Elton R. Yasuna and Leah Sanders Green 235
Menière's disease associated with traumatic hyphema and glaucoma. Harry McGrath 241
LeConte's book on "sight." Beulah Cushman 245
Trichlorethylene as an analgesic in ophthalmic practice. Bertha Offenbach 250
True polycoria. Norman S. Jaffe and Paul Knie 253

NOTES, CASES, INSTRUMENTS

Moebius's syndrome: Congenital oculofacial paralysis: A case report. J. R. Stansbury 256
Paradoxal ocular motility after cycloplegia. James Miles O'Brien 261

SOCIETY PROCEEDINGS

College of Physicians of Philadelphia, Section on Ophthalmology, March 15, 1951 263
New York Society for Clinical Ophthalmology, February 5, 1951 264

EDITORIALS

IV Pan-American Congress of Ophthalmology 268
Specialist examinations in Canada 270

CORRESPONDENCE

Ocular manifestations of some tropical diseases 272
Research grants available 275

BOOK REVIEWS

Genetics in Ophthalmology 276
Ophthalmology 276
Chronology of Ophthalmic Development 277
Bulletin of the Belgian Ophthalmological Society 277
Transactions of the Ophthalmological Society of Tuscany, Umbria, and Emilia 280

ABSTRACTS

General pathology, bacteriology, immunology; Vegetative physiology, biochemistry, pharmacology, toxicology; Physiologic optics, refraction, color vision; Diagnosis and therapy; Ocular motility; Conjunctiva, cornea, sclera; Uvea, sympathetic disease, aqueous; Glaucoma and ocular tension; Crystalline lens; Retina and vitreous; Neuro-ophthalmology; Eyeball, orbit, sinuses; Eyelids, lacrimal apparatus 281

NEWS ITEMS 298

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AMERICAN JOURNAL OF OPHTHALMOLOGY

VOLUME 35 FEBRUARY, 1952 NUMBER 2

THE EFFECT OF SUTURES AND OF THROMBIN UPON OCULAR WOUND HEALING*

THE JACKSON MEMORIAL LECTURE

JOHN H. DUNNINGTON, M.D., AND ELLEN F. REGAN, M.D.
New York

This lecture established in the memory of Dr. Edward Jackson is a most fitting tribute to him whose untiring efforts did so much to establish this academy's preeminence in the educational world. He was an inspiring teacher whose clear thinking and sympathetic understanding endeared him to all. He was a tireless worker for the cause of ophthalmology, and this society—the object of his love—profited greatly from his sage advice and boundless enthusiasm. I am most appreciative of this honor for it gives me the opportunity to express publicly my great admiration for this distinguished ophthalmologist. His was a practical mind so in this presentation some clinical aspects of ocular wound healing will be emphasized.

GENERAL PRINCIPLES OF WOUND HEALING

Before discussing the behavior of the reparative processes in ocular incisions it is appropriate to review two aspects of wound healing which have been investigated extensively by the general surgeons. We shall not attempt to cover the entire subject but the methods of repair and the effect of sutures are, for purposes of orientation, worthy of consideration.

1. *Method of repair.* From 1916 to 1920, Spain and Loeb,[1,2] in reporting the cellular changes that occur in the course of normal healing, stressed the importance of amoeboid movement, mitotic proliferation, and the maturation of the cells concerned in the fusion of wound surfaces. They demonstrated that there is a latent period of three to five days during which the lips of the wound are held together insecurely by a loose fibrin meshwork. This is followed by contraction of the meshwork and proliferation and maturation of fibroblasts, thus producing a firmly healed wound in 12 to 14 days. The importance of this fibroblastic response was emphasized by Samuel Harvey[3] in 1929, when he stated that it is the master reaction in cicatrix formation. Later Christopher[4] showed that this fibroblastic tissue is not so strong as normal tissue and hence a broad area of scar is not desirable.

2. *Effect of sutures.* Although many suture materials have been employed in general surgery, our attention has been directed to those commonly used by ophthalmologists —that is, silk and catgut. While all investigators are agreed that sutures are essential to hold the edges of the wound in contact until fibroblastic proliferation has been established,[5] it is admitted that the effectiveness of a suture may vary considerably.

Among the factors which influence them are: (1) The duration of tensile strength,

* From the Department of Ophthalmology of Columbia University and The Institute of Ophthalmology of Presbyterian Hospital. Presented at the 56th annual meeting of the American Academy of Ophthalmology and Otolaryngology, Chicago, October, 1951.

(2) the tissue response incited by the suture material, (3) the rate at which it is absorbed, and (4) the degree to which it predisposes to infection.

According to most studies, silk sutures maintain their tensile strength for about seven days and incite only a moderate amount of round cell infiltration.[6, 7] The disadvantages of this material are: (1) It is not absorbable, (2) it has a greater tendency to cut through tissue than does catgut, and (3) small shreds remaining after removal may act as a nidus of infection.[8]

A plain catgut suture has been shown to lose its tensile strength in about four days and to be absorbed in seven days.[8, 9] Jenkins and his co-workers[10] have demonstrated that plain catgut produces a rapid and intense polymorphonuclear infiltration. Howes and Harvey[8] believe this material delays fibroblastic proliferation. These investigators[8] also found that surface wounds when closed with catgut are prone to develop infection.

In contrast, chromic catgut sutures generally retain their tensile strength for five to six days and are absorbed in 21 days.[10] Both the tensile strength and the rate of absorption of all catgut materials are extremely variable. In the presence of extensive cellular reaction or infection even chromic catgut sutures may absorb completely within four or five days, while, if the response is minimal, they may persist well over their accredited 21 days.[10, 11]

It is of interest to note that, contrary to the usual concept held by ophthalmologists, several investigators have reported that chromic catgut sutures incite less reaction than do plain catgut. Among them is Farris[12] who placed pieces of various suture materials in the anterior chambers of rabbits and studied the reactions produced by them. He found that there was very little cellular response to silk, considerably more to chromic gut, and that an even greater amount of reaction occurred when plain gut was left in the anterior chamber of the rabbit's eye.

Jenkins[10] has demonstrated that chromic catgut sutures initially incite a very limited polymorphonuclear response which is followed in seven to 10 days by the invasion of phagocytic cells. Therefore, it would appear that, although plain catgut produces a more severe cellular reaction, it is of shorter duration than that caused by chromic catgut.

Ocular wound healing

In view of these established facts on wound healing and recognizing the paucity of our knowledge of the methods of repair of ocular wounds we began this study. Although the preliminary results of these investigations have been presented,[13] a brief review of some of our findings is pertinent. We first considered the normal methods of healing of cataract incisions with and without conjunctival flaps.

From our experimental studies on a series of monkeys and from Purtscher's[14] and Maggiore's[15] investigations it appears that healing occurs first anteriorly and gradually progresses posteriorly. In the presence of a conjunctival flap the initial closure takes place through the formation of a fibrovascular plug of episcleral tissue. This fills the anterior one half of the incision by the end of the second day, while the first evidence of endothelial proliferation is not seen until the fifth or sixth day.

If the corneal incision is used, the primary closure is effected by an epithelial plug formed by a proliferation of the corneal epithelium. This first bridges the break in the anterior surface and then extends down along the lips of the wound wherever the edges are not in close approximation.

Under normal conditions healing by either of these methods is firm in 10 to 12 days and is complete by the end of the third week.

Then turning our attention to the abnormalities of wound healing, we found that minor imperfections usually result from (1) overlapping, (2) anterior gaping, and (3) posterior gaping.[12] These variations from the normal lead to less firm closure of the incision. In the absence of a conjunctival

flap either overlapping or anterior gaping produces excessive proliferation of corneal epithelium with the formation of a large anterior epithelial plug, while if a similar situation exists under a flap there is an increased formation of fibrovascular tissue derived from the episcleral structures. Gaping of the posterior part of the incision increases the chances of tissue inclusion.

Effect of sutures

Having demonstrated the mechanisms of repair in the presence of faulty wound closure, we then became interested in the cause of these imperfections.

In view of the almost universal use of corneoscleral sutures, the effect they exert on healing was studied. In all of our experiments the Rhesus monkey (macaca mulatta) was used and the cataract incisions were made either with the Graefe knife or with the keratome and scissors. Black silk (6-0) and plain catgut (5-0) were the suture materials used. Two appositional postsectional corneoscleral sutures were employed along with a sufficient number of supplementary conjunctival ones to close the wound adequately.

Fig. 1 (Dunnington and Regan). Eye of a monkey two days after operation, showing (A) epithelium growing along track of suture.

The animals were killed at varying intervals over a four-month period and the eyes were sectioned serially. Microscopic study of the entire length of the incision was made, paying particular attention to the reactions around the corneoscleral sutures. In these areas several interesting and significant happenings take place.

First, there is a proliferation of the conjunctival or corneal epithelium along the track of the suture. In some instances this occurs with such rapidity that the entire path of the suture is completely outlined by epithelium at the end of two or three days (fig. 1).

Second, the area around the suture shows small round-cell infiltration of varying degree.

Third, stromal necrosis and decreased fibroblastic proliferation are noted in this area. The extent of the necrosis depends upon the amount of tissue incarcerated by the suture and the type of material used. If the suture is not too deeply placed it will usually slough out within two or three weeks, leaving a furrow whose extent depends upon the depth at which the suture was inserted (fig. 2). Such reactions are usually microscopic in character and often appear in wounds which to casual inspection have healed perfectly.

The pendulum of surgical thought having swung in recent years toward deep firm sutures, we next deliberately placed many in this manner. Clinically, the incisions appeared to have healed well, the only abnormality being the occasional appearance of a slightly updrawn pupil or an incarcerated iris pillar. Histologically, however, these wounds showed tissue reactions sufficiently

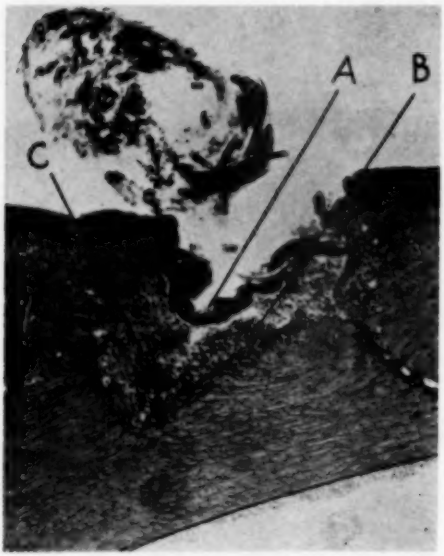

Fig. 2 (Dunnington and Regan). Eye of a monkey three weeks after operation, showing reaction around suture. (A) Epithelized furrow, (B) mild stromal necrosis, (C) slight round-cell infiltration.

severe to warrant serious consideration.

The epithelial proliferation along the track of the suture in a few instances extended into the anterior chamber (fig. 3). The stromal necrosis was extensive and was surrounded by marked round-cell infiltration with little or no evidence of fibroblastic repair (fig. 4). When this reaction was intense even the epithelium about the suture degenerated and the edges of the incision were then bound together only by a few strands of fibrous tissue surrounded by round cells and necrotic tissue. In the part of the wound adjacent to the suture there was anterior gaping of the incision with increased formation of highly vascularized fibroblastic tissue.

The following clinical conplications could result from such abnormalities in healing:

1. *Epithelization of the anterior chamber.* In 1939, Terry, Chisholm, and Schonberg,[16] in an article on the invasive quality of surface epithelium, mention a slide obtained by Verhoeff in 1901 showing epithelial proliferation along a scleral stitch. These observers along with others[17] have long been conscious of this tendency of the epithelium to follow the course of a suture. From this knowledge it has been agreed that epithelium could gain access to the interior eye if a penetrating suture is left in place long enough.

In Figure 3 such an ingrowth of epithelium is visible within the anterior chamber of a monkey's eye. The operative procedure on this eye varied in no detail from that used on others except that two penetrating corneoscleral sutures were inserted and remained in place for three weeks at which time the animal was killed. No undue postoperative reaction occurred nor was there any appreciable delay in reformation of the anterior chamber.

One can at least theorize that the suture

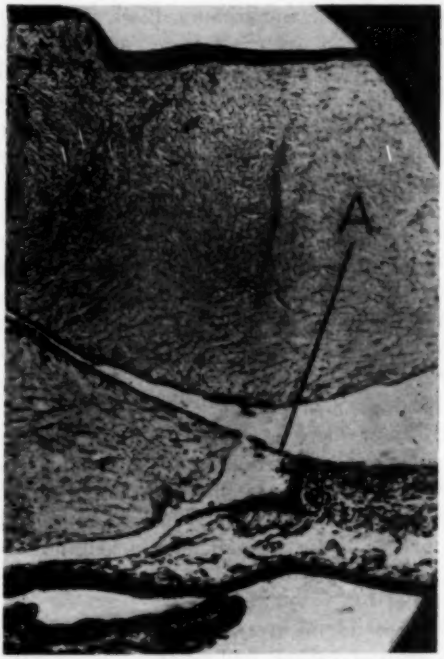

Fig. 3 (Dunnington and Regan). Eye of a monkey two months after operation, showing (A) epithelium invading anterior chamber along the track of a deep suture.

Fig. 4 (Dunnington and Regan). Eye of a monkey three weeks after operation, showing (A) extensive stromal necrosis, (B) marked round-cell infiltration with absence of fibroblastic repair.

need not penetrate because, if it is sufficiently deeply placed to cause loss of most of the overlying stroma, subsequent wound rupture could allow the invading epithelium to reach the interior of the eye.

2. *Fistulization.* From our present investigations it appears that, in the vicinity of a deeply placed suture, histologic changes occur that are ideal for the formation of a fistula. If this type of suture is allowed to remain in place too long, the resulting tissue necrosis, the round-cell infiltration, and the decrease in fibroblastic response will produce a weakened area in the wound (fig. 4). The necrotic material will slough out and, if this loss of substance extends deep enough, fistulization may occur.

Since such an area has devitalized edges, it will not heal readily or firmly and may be expected to break down repeatedly. This occurrence would explain the intermittent nature of the clinical symptoms associated with a small fistulizing cicatrix.[18] When the track of the suture becomes completely lined with epithelium the fistula can be expected to remain open.

These histologic changes indicate why cautery or curettage of the wound edges is an essential part of any operation designed to obliterate permanently such a fistula.

3. *Delayed iris incarceration or prolapse.* The probable sequence of events leading to either of these conditions is as follows: A firmly tied, deeply placed suture has caused

Fig. 5 (Dunnington and Regan). Eye of a monkey two months after operation, showing (A) slough, (B) adherent iris in region of deep sutures.

necrosis of the tissue incorporated in the bite.[19] The resultant slough which extends almost through the cornea and sclera has led to the formation of a thin or weakened spot in the wound. With the return of the ocular tension to normal this weakened area bulges or gapes, thus causing the iris to become adherent in this region (fig. 5).

When the incarcerated uveal tissue is small in amount but adequate to prevent approximation of the deeper layers a cystoid cicatrix is the expected result; whereas, when a larger amount of iris lies between lips of the wound, a frank prolapse usually develops.[13]

4. *Hemorrhage into the anterior chamber.* It is generally accepted that most hemorrhages into the anterior chamber arise from the newly formed capillaries which invade the edges of the incision.[20] In any abnormality of wound healing where the method of repair is effected chiefly by the formation of a large anterior fibrovascular plug, hemorrhage is more prone to occur.

Such a condition is seen when histologic examination is made of the wound areas adjacent to firmly tied, deeply placed corneoscleral sutures. Close approximation of the tissues included in the bite may produce an anterior gaping of the intervening areas as shown in Figure 6.

The presence of the large number of blood vessels in this area may well account for the frequency with which hemorrhage into the anterior chamber occurs in wounds closed with deep sutures. Such hemorrhages may occur either spontaneously or at the time of removal of the suture.

Effect of thrombin

In an attempt to find a substitute for sutures, our attention turned to the use of fibrin

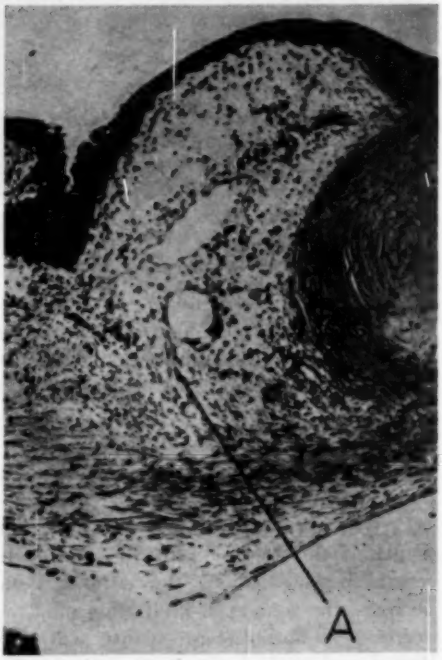

Fig. 6 (Dunnington and Regan). Eye of a monkey one month after operation, showing (A) large vascularized anterior plug as a result of improper approximation of wound in area between deep sutures.

formed by the combination of thrombin and fibrinogen. This method was first used to attach free skin grafts by Cronkite, Lozner, and Deaver.[21] Young and Medawar[22] used a similar procedure for suturing nerves, and Young with Favata[23] reported the successful use of thrombin and plasma in the closure of traumatic lacerations.

From these reports it would appear that this technique has the advantage of producing a rapid agglutination of the wound edges followed by normal healing and avoids the reaction produced about sutures. The disadvantage lies in the fact that fibrin has a low tensile strength and consequently is unable to preserve closure of a wound upon which any tension is exerted.

Recently fibrin has been suggested as a substitute for sutures in ophthalmic surgery. In 1946, Brown and Nantz[24] showed experimentally that fibrin increases the adhesive power of a corneal wound.

Later Tassman[25] applied these principles to the closure of cataract incisions. He used no sutures but applied plasma and thrombin along the edges of the wound, and then held the lips of the wound in apposition until the fibrin coagulum became fixed. Town and Naidoff,[26] who used a similar technique, stated that the use of fibrin alone for the closure of cataract incisions reduces the number of postoperative leaky wounds and lessens the incidence of hyphema, epithelial downgrowth, and iris prolapse.

Tassman[27] has also advocated the use of fibrin to seal corneal grafts and both he and Byrnes believe it is indicated in the repair of leaking wounds and corneal lacerations.[26, 28]

In England, Parry and Laszlo[29] and in Budapest, von Grosz[30] have advised using fibrin to close cataract incisions.

In order to evaluate these findings, cataract incisions similar to those used in the previous experiments were made on monkeys. Two or three drops of a solution containing 500 units per cc. of thrombin in normal saline were applied either to the surface of the corneal wound or beneath the conjunctival flap. An equal amount of lyophilized plasma was added immediately and the edges of the wound were held together for about one minute. No sutures were employed. The animals were killed at 2, 4, 7, 14, and 21 days postoperatively and the eyes sectioned serially.

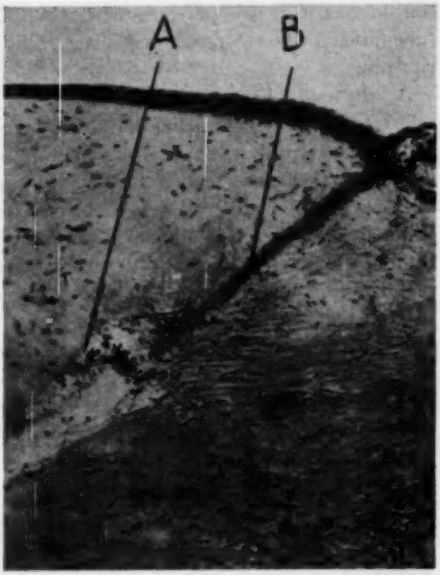

Fig. 7 (Dunnington and Regan). Eye of a monkey two days after operation, showing (A) absence of cellular response, (B) proliferation of epithelium along the edges of a corneal incision to which thrombin was applied.

Histologic examination at the end of two days showed fibrin deposits beneath the conjunctiva in cases in which a flap was used. Irrespective of the use of a flap, fibrin was present between the wound edges, producing poor approximation. There was no evidence of fibroplastic proliferation and, in some instances, epithelium was seen growing down between the lips of the wound (fig. 7).

At the end of four days, some fibroblasts were seen in the vicinity of the incision, there was little or no round-cell infiltration, and the epithelium had proliferated still further along the wound. When a

conjunctival flap was present, the fibrin and subepithelial tissues constituted a large anterior plug.

At the end of one week fibroblasts had infiltrated this subepithelial tissue and invaded the fibrinous meshwork, making a broad scar which extended throughout the entire wound (fig. 8). When a corneal incision was used, the epithelial downgrowth sometimes lined both edges of the wound throughout its entire length. Fibroblasts were present beneath the epithelium (fig. 9).

After two weeks, all incisions seemed well healed with dense broad fibrous cicatrices, the only exception being in those eyes in which the edges of the corneal wound were epithelized.

From these studies we believe that, in the presence of fibrin, normal healing will occur without the round-cell invasion or necrosis found in the vicinity of sutures. During the first few days of healing fibrin tends to separate the edges of the wound. This may produce three undesirable effects: (1) The cohesive action of properly approximated wound edges is lacking; (2) a broad fibrous cicatrix will be created which, although usually satisfactory, will lack the tensile strength of normal tissue; (3) as suggested by Brown and Nantz,[31] in the presence of a corneal incision epithelium may proliferate along the lips of the wound, producing a weakened area or possibly epithelization of the anterior chamber.

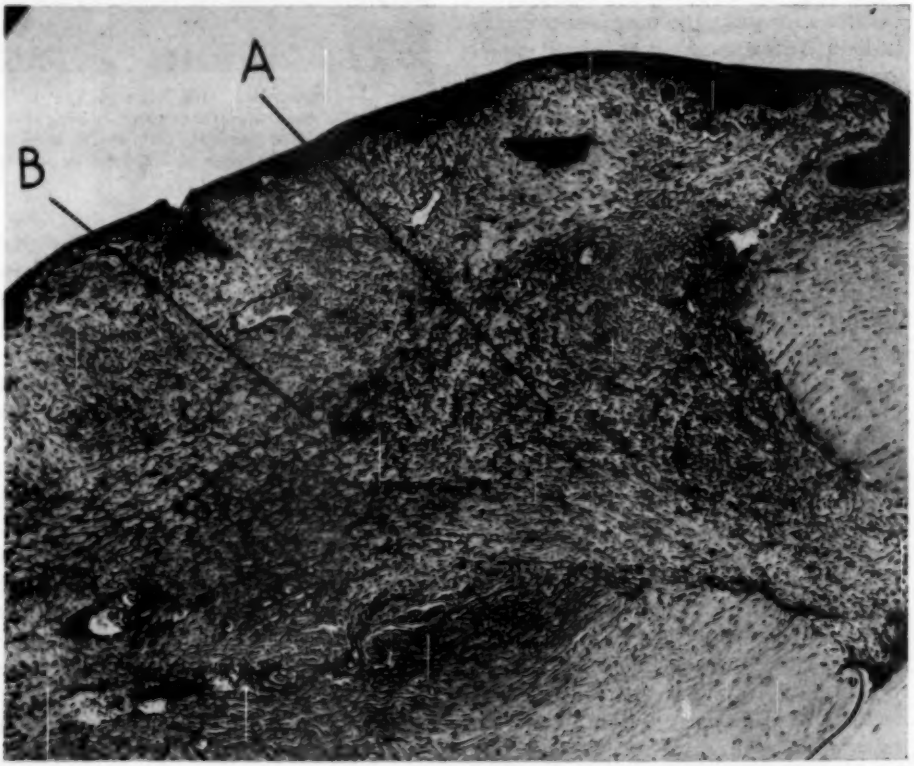

Fig. 8 (Dunnington and Regan). Eye of a monkey four days after operation, showing an incision in which fibrin was applied under the conjunctival flap. (A) Large anterior plug of fibrin and subepithelial tissues, (B) separation of edges of wound.

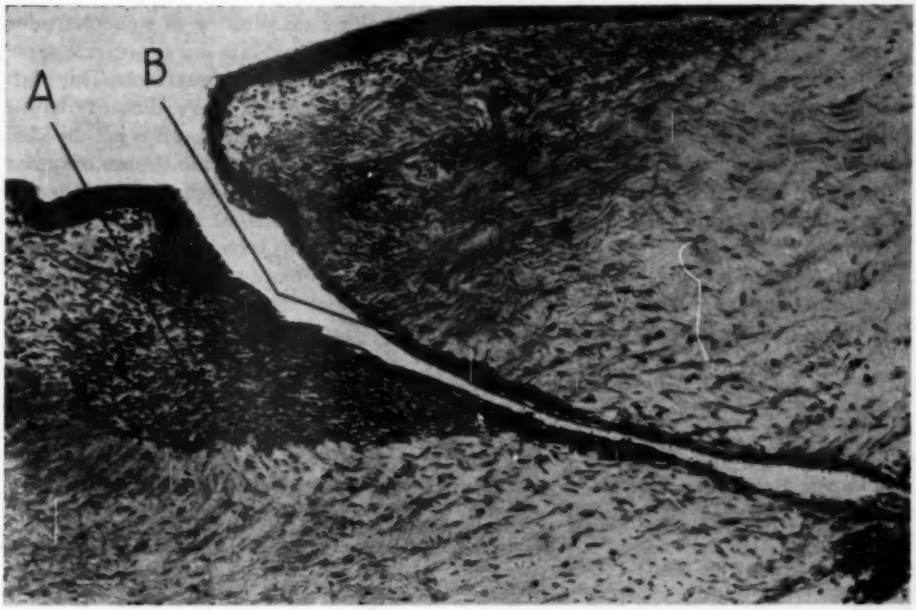

Fig. 9 (Dunnington and Regan). Eye of a monkey seven days after operation, showing a corneal incision to which fibrin was applied. (A) Subepithelial fibroblastic proliferation, (B) epithelial downgrowth along edges of wound.

Summary

The principles of wound healing in general have been discussed because it is felt that the importance of the latent period in healing it not fully appreciated by ophthalmic surgeons. Histologic examination of ocular wounds shows that normal healing occurs first on the anterior surface and progresses posteriorly. During the first three days, the repair of corneal incisions is limited to epithelial proliferation; while in wounds with a conjunctival flap this anterior closure is effected by fibroblastic proliferation. Under abnormal conditions these reparative processes may become so exaggerated that defective closure results.

Based upon these histologic studies, it is apparent that, to insure normal healing, ocular incisions should be in apposition during the latent period. In an attempt to study the ways and means of achieving such apposition, sutures at varying depths were used and the reactions around them examined microscopically.

Superficially placed sutures were found to produce gaping of the posterior half of the incision, a condition which predisposes to tissue inclusion. Deep sutures incite severe reactions, particularly when left in place for too long a period.

Chief among the microscopic changes that occur around deeply placed sutures are: (1) Rapid proliferation of the epithelium along the track of the suture, (2) extensive necrosis of the tissues included in the bite, (3) excessive formation of highly vascularized fibroblastic tissue in the adjacent portions of the wound.

The complications that may result from these reactions are: (1) Epithelial invasion of the anterior chamber, (2) fistulization, (3) iris incarceration or prolapse, (4) hemorrhage into the anterior chamber.

From these histologic studies it would

appear that a corneoscleral suture should not be inserted to a depth greater than one half the depth of the incision. Because of the foreign-body reaction sutures incite, their number should be limited and their removal not delayed beyond 12 days.

Inasmuch as the tensile strength and rate of absorption of catgut vary with the degree of reaction, it is felt that silk is preferable, although further investigations with mildly chromicized gut as advocated by Fralick[19] may prove it to be the material of choice.

Histologic examination of wounds treated with thrombin indicate that it prevents apposition of the wound surfaces. This fact combined with its very limited tensile strength makes us feel that it is not the ideal material to insure accurate closure of ocular wounds. These microscopic studies also confirm the usefulness of a conjunctival flap, for wounds with such a covering heal more promptly and more securely than do corneal incisions.

635 West 165th Street (32).

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LENS-INDUCED UVEITIS AND GLAUCOMA*

Part I. Endophthalmitis phaco-anaphylactica

S. Rodman Irvine, M.D., and Alexander Ray Irvine, Jr., M.D.
Beverly Hills, California

It is generally recognized that the presence of disorganized lens tissue in the eye may result in insignificant reaction, or may result in various types and grades of inflammation which subside when the lens or lens substance is removed.

The types of inflammatory response and their clinical prototypes, alleged to pertain to the lens, have been described a number of times. Excluding the glaucoma caused by blocking of the iridocorneal angle by an intumescent lens, we find in the literature:

I. *The phaco-anaphylactic response,* described by Verhoeff and Lamoine,[1] Burky,[2] Heath,[3] and Courtney.[4]

II. *The so-called toxic response,* described by Verhoeff,[1] Gifford,[5] Knapp,[6] and Heath.[3]

III. *"Phaco-genetic" glaucoma,* in which the angle meshwork is obstructed by large eosinophilic phagocytes, as demonstrated by Zeeman.[7]

Recent clinical reports of the reaction of the tissues of the eye to lens substance, and to cataracts, have been presented by Irvine and Irvine,[8] Hughes and Owens,[9,10] and Sugar.[11]

Notwithstanding these reports in the literature, eyes are received in every eye pathology laboratory, showing anterior segments with pathologic changes but relatively normal posterior segments, suggesting that these eyes might have been saved if the lenses had been removed. These eyes, no doubt, were enucleated either because, clinically, they seemed hopelessly lost (there being faulty light projection) or because of the fear of ensuing sympathetic ophthalmia.

When inflammation occurs in an eye after injury or surgery to the opposite eye, the inflammation is often considered to be sympathetic uveitis. One can readily appreciate the surgeon's hesitancy in operating upon either eye in such cases, a hesitancy prompted, very likely, by the catastrophic results that follow surgery in cases of uveitis in general, and particularly in cases of sympathetic ophthalmia.

Our data presented in this paper further classifies information with regard to the types of inflammation associated with pathologic conditions of the lens, and further emphasizes the clinical differentiation from other types of uveitis of the curable condition of lens-induced uveitis with glaucoma.

Of the eyes received at the Estelle Doheny Eye Laboratory during the past two years, 128 showed injury to the lens following trauma or operation, and 12 of these showed inflammation believed to be caused by the lens substance. Sixty-five showed insignifi-

* From the Estelle Doheny Eye Foundation Laboratory, Los Angeles, California. Presented before the Western Section of the Association for Research in Ophthalmology, Portland, Oregon, 1950.

cant inflammation in or about the lens tissue. In 51 eyes, the inflammation was so extensive as to obscure any part played by the lens.

This report includes 11 injured eyes with inflammation believed to be related to the lens (Cases 1, 2, 3, 4, 5, 6, 7, 8, 10, 11, and 18), four additional eyes in which inflammation arose spontaneously (Cases 1, 9, 13, and 19), five clinical cases (Cases 12, 14, 15, 16, and 17) in which pathologic material never became available, and one case of sympathetic ophthalmia with coincidental lens-induced uveitis (Case 20).

Of the 15 eyes examined pathologically and believed to show reaction to lens matter, some were hopelessly lost for other reasons than the inflammation related to the lens, but nine might have been saved if the lens had been removed. For purposes of description the types of response in the 20 cases reviewed have been divided into the three groups mentioned in the literature.

I. Endophthalmitis phaco-anaphylactica

(Cases 1 through 8 and Case 20)

The importance of the phaco-anaphylactic element in initiating inflammation in the eye is controversial (Braun[12]) but, although lens substance is a weak antigen (Woods and Burky[13]), there is evidence that certain reactions have an allergic aspect (Verhoeff and Lemoine,[14] Burky,[16] Burky and Henton,[17] Gifford,[18] and, histologically, there is a group, the characteristics of which were first stated by Verhoeff.[1]

These characteristics include:

1. Invasion of the lens by polymorphonuclear leukocytes and mononuclear phagocytes.

2. The presence of giant cells and polymorphonuclear cells about the lens fragments, in the iris, and in the pupillary membrane which often forms.

3. The deposition of conglomerate precipitates on Descemet's membrane with, sometimes, proliferation of a syncytial connective tissue in the anterior chamber in which various types of inflammatory cells can be found, including giant cells, polymorphonuclear cells, and occasionally eosinophils.

4. The presence of a cyclitic membrane in protracted cases.

5. A relatively unaffected posterior segment. Lens fragments in the vitreous may cause a collection of pus cells and macrophages in the vitreous, as well as collections of cells on the retina, similar in formation to keratic precipitates. The pathognomonic finding is the combination of polymorphonuclear leukocytes, and macrophages and giant cells phagocytizing lens material.

This group has been adequately described by Verhoeff, Heath, and others, both as to the clinical course and the pathologic findings. We present additional cases further to emphasize points they already have suggested, and as an introduction to the discussion of cases in Groups II and III.

A typical case follows:

Case 1

January 9, 1946. A 90-year-old women was seen for the first time on this date.

Her right eye was quiet but it had been blind for approximately a year, reportedly from uveitis and glaucoma. The cornea showed bullous keratitis with marked opacity of the endothelium on which there were large mutton-fat keratic precipitates. The iris was heavily vascularized. The pupil was semidilated and fixed. The eye was hard. A mature cataract was present, but this is not described in detail.

The left eye, on this date, showed a narrow-angle glaucoma; tension of 30 to 40 mm. Hg (Schiøtz); no gross defect by confrontation fields; vision, 20/200. The tension could not be controlled with miotics.

May 20, 1947. After an acute rise in tension, iridectomy was performed. Following keratome incision at the limbus, the iris prolapsed spontaneously so that it could be grasped without entering the anterior chamber. A basal iridectomy was executed. The

operation was complicated by slight hyphema.

The cornea remained hazy, and there appeared to be a slight exudate on the lens. Tension remained a high normal, a marked aqueous flare persisted, and the lens became completely opaque. Approximately one month after surgery fine keratic precipitates could be seen developing. These increased and became massive conglomerate keratic precipitates covering the entire cornea. Tension increased to 50 mm. Hg.

June 23, 1947. The blind right eye was removed for diagnosis.

July 11, 1947. As the findings on the right eye indicated a diagnosis of reaction to lens matter, an aqueous puncture was made on the left eye to obtain confirmatory evidence; 0.2 cc. of aqueous were aspirated and the total protein, by Pandy test, was estimated as approximately 2000 mg. per 100 cc. On standing, no clot formed in the fluid.

The remaining fluid was dried on a slide and stained with Wright's stain. The cells were very few, there being no clumps of cells and only a rare lymphocyte, monocyte, and polymorphonuclear cell. No eosinophils were found. There was considerable amorphous material present.

A diagnosis of reaction to lens material was made notwithstanding a negative skin test to an emulsion of human cataractous lens.

July 15, 1947. A cataract extraction was performed. Following the section, the lens was slid out intracapsularly without loss of vitreous. Grossly, the capsule was intact. The lens was a large, soft, mature cataract.

In spite of a bulging vitreous body immediately after delivery of the lens, no anterior synechias developed and the post operative course was surprisingly good. All signs of uveitis subsided. All the keratic precipitates disappeared.

October 10, 1947. Three months after operation, the eye was white and quiet, the cornea clear, the anterior chamber normal in depth, and the vitreous face intact. Vision, with a plus 15D. lens, was 20/70.

March 15, 1948. There were still no signs of uveitis, tension was normal, and vision was 20/40. The pathologic report on the enucleated right eye follows.

Description of pathologic anatomy

There is marked perilimbal, lymphocytic, and plasmocytic inflammation. Small blood vessels within the epithelium and beneath it at the limbus at one side suggest an early degenerative pannus. Blood vessels are seen in the stroma peripherally.

An organized fibrotic syncytium lines the endothelium, chamber angles, anterior (fig. 1) surface of the iris, verging on the pupil, thus filling the anterior chamber except for an oval space centrally measuring five mm. transversely. This loose syncytium is comprised of mature connective tissue cells with spindle-shaped nuclei and long interlacing protoplasmic processes, probably derived from the endothelium by metaplasia.

Many polymorphonuclear leukocytes and giant cells (figs. 2 and 3), both of the Langhan's and foreign body type, are scattered through it. Small accumulations of polymorphonuclear leukocytes and mononuclear inflammatory cells, and occasionally giant cells, line the inner surface of this membrane, sug-

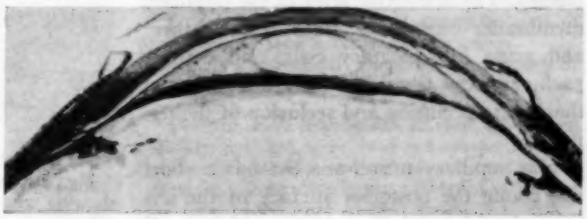

Fig. 1 (Irvine and Irvine). *Case 1.* Infiltrate of iris and syncytium in anterior chamber.

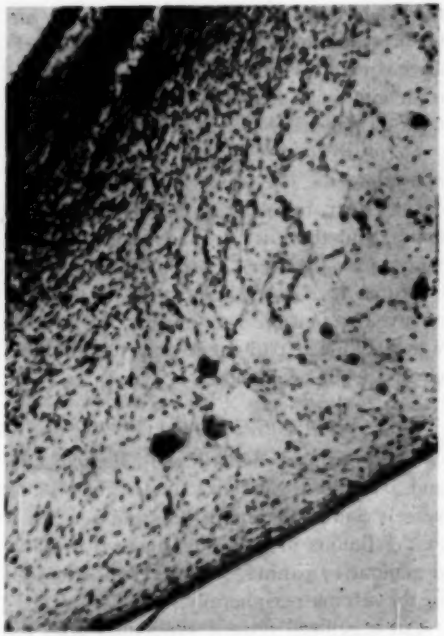

Fig. 2 (Irvine and Irvine). *Case 1.* Syncytium showing giant cells. (Low-power view.)

gesting that aqueous circulated through the oval space already described.

There are dense peripheral anterior synechias. The iris root is markedly compressed and thinned. The pigment epithelium is thinned and atrophic and is reflected posteriorly at the edge of a dense pupillary membrane. The architecture of the iris stroma has been greatly altered and blends imperceptibly into the connective-tissue membrane in the anterior chamber. The stroma is heavily infiltrated with lymphocytes and plasma cells.

Unfortunately the lens was lost in sectioning. The dense character of the pupillary membrane, containing giant cells, chronic and acute inflammatory cells, and densely packed fibrous tissue elements, suggests that there were occlusion and seclusion of the pupil.

The pupillary membrane extends a short way along the posterior surface of the iris on each side, and the presence of a small fragment of lens capsule and lens cortex in its posterior margin suggests that it was tightly adherent to the lens.

Some of the lens fragments showed the presence of capsular epithelium, whereas other fragments did not. There was an occasional polymorphonuclear cell seen in the lens substance in some sections.

There is extreme atrophy of the ciliary body. The epithelium is flattened; its processes thinned and hyalinized. The nerve-fiber layer of the retina is gliosed, ganglion cells are not evident, the optic nerve is sharply, though shallowly cupped, and the nerve trunk is atrophic and gliosed.

Impression. Endophthalmitis phaco-anaphylactica. Secondary absolute glaucoma.

Comment. The inflammatory syncytium in the anterior chamber, containing polymorphonuclear cells and giant cells, plus the heavy infiltration of the iris with plasma cells and lymphocytes simulate findings described

Fig. 3 (Irvine and Irvine). *Case 1.* Syncytium showing giant cells and polymorphonuclear cells (High-power view.)

by Verhoeff in some of his cases of endophthalmitis phaco-anaphylactica.

The paucity of inflammatory changes in the posterior segment is noteworthy. The absence of tubercle-forming epithelioid cells distinguishes the reaction from chronic granulomatous inflammation.

The sections were not stained for fungi and, although the combination of giant cells and polymorphonuclear cells is also characteristic of infections by fungi, the history and the diffuseness of the process is inconsistent with such a diagnosis.

Comment on Group I

We believe that there is evidence, pathologically, that the patient whose case is reported in detail had lost vision in the right eye from uveitis resulting from inflammation caused by the presence of lens material. The same course of events was certainly progressing in the left eye, and it seemed hopelessly involved until miraculously cured by removal of the lens.

The synopses of Cases 2 through 8 and Case 20, which fall into the same category as Case 1, are appended.

Cases 1 through 8 and Case 20 show the presence of polymorphonuclear leukocytes and giant cells in the inflammation around the lens, or lens fragments, except Case 7 which shows no giant cells, and Case 8 which shows no polymorphonuclear cells. Reference will be made to these again, in the analysis of Group II. In five cases (2, 3, 5, 6, and 8) the posterior segment was relatively normal.

It is interesting that in the cases in Group I, the lens was cataractous, either from ordinary senile change or from injury, but that the lens cortex was not completely broken down as it is in the hypermature state—this was judged by the finding of normal epithelium in conjunction with lens fragments. When the lens cortex is liquefied, normal epithelium is not present.

We were unable to correlate the presence or absence of giant cells, polymorphonuclear leukocytes or eosinophils, with the degree of degeneration or dissolution of the lens substance or the duration of the process.

Synopses of cases in Group I*

Case 2

December 16, 1947. A woman, aged 72 years, had an uneventful intracapsular cataract extraction of the right eye, resulting in corrected visual acuity of 20/20.

September 15, 1948. A cataract extraction was performed on the left eye with loss of fluid vitreous. The lens was dislocated into the vitreous and removed by loop. Postoperative inflammation developed.

December 15, 1948. The left eye was removed, since the inflammation would not subside. The right eye at this time showed no signs of inflammation.

Description of pathologic anatomy

Microscopically there is moderate perilimbal infiltration. The anterior chamber is open except nasally where there is a loosely adherent peripheral synechia just occluding the trabecular meshwork. A few lymphocytes, plasma cells, and mononuclear phagocytes, some containing pigment, are seen within the sclerosed trabecular meshwork and along the endothelium.

The iris stroma is somewhat compressed, fibrosed and atrophic. The normal crypts are not apparent. There is moderate atrophy of the pigment epithelium, and a normal ruffled appearance is not seen. A dense organized pupillary membrane bridges the iris leaves and binds them to cataractous lens remnants posteriorly. Lymphocytes, plasma cells, and "clump cells" are scattered diffusely throughout the iris stroma. Their concentration is greatest at the pupillary portion and within the pupillary membrane.

Cataractous lens remnants having normal epithelium are seen behind the iris and pupillary membrane. The cortex is invaded by mononuclear phagocytes, plasma cells, lymphocytes, and occasionally giant cells of both the foreign-body and Langhan's type. Of particular interest is the presence of small foci of necrotic polymorphonuclear leukocytes. A moderate number of eosinophilic leukocytes are seen just posterior to the capsule within the organizing lens cortex, and behind the pupillary membrane.

Mononuclear phagocytes and chronic inflammatory cells are scattered through the anterior vitreous, and along the surface of the pars plana and retina, as far back as the equator. Occasionally nests of phagocytes are seen in the posterior vitreous.

There is a perivascular lymphocytic infiltration of the nerve-fiber layer of the retina adjacent to the

* For pathologic examination all specimens, unless otherwise specified, were fixed in formalin Zenkers, embedded in celloidin, and stained with hematoxylin and eosin. Gross descriptions, recorded when the eyes were opened, are omitted unless pertinent.

disc. Ganglion cells are abundant. There is a small flat hemorrhagic separation of the retina nasally extending from the disc a distance of about five mm.

The choroid is remarkably free from inflammation, but free blood is seen in the suprachoroidal space anteriorly.

Diagnosis. Secondary cataractous membrane; endophthalmitis phaco-anaphylactica; serous retinitis; hemorrhagic detachment of choroid following operation.

Comment. The presence of giant cells, polymorphonuclear leukocytes, eosinophilic leukocytes, and macrophages with their concentration in the iris and about the lens remnants, is of particular significance. The absence of tubercle-forming epithelioid cells distinguishes this reaction from a chronic granulomatous inflammation.

CASE 3

July, 1947. A woman, aged 72 years, had a cataract extraction of the right eye. Postoperative uveitis and glaucoma developed. The left eye developed uveitis diagnosed as sympathetic ophthalmia.

July, 1948. The right eye was enucleated.

Description of pathologic anatomy

Microscopically, the corneal epithelium is irregular, edematous, and contains intraepithelial bullae. Nasally there is newly formed connective tissue between Bowman's membrane and the overlying epithelium. A few blood vessels are seen throughout the deeper layers of the stroma centrally and temporally.

A healed penetrating wound of the cornea is seen about two mm. inside the limbus temporally. The endothelium is flattened and attenuated.

There are broad peripheral anterior synechias occluding the chamber angle. The anterior chamber is filled with serum and contains a few chronic inflammatory cells and phagocytes.

The iris root is compressed, fibrotic, and atrophic. The pupillary irises are densely infiltrated with lymphocytes and plasma cells. There is atrophy of the pigment epithelium, and pigmented "clump cells" are seen within the stroma.

An organized inflammatory membrane occluding and secluding the pupil attaches posteriorly to a cyclitic membrane in which cataractous lens remnants are evident.

Lens epithelium is seen nasally. The cyclitic membrane probably derives its connective tissue from metaplastic lens and ciliary epithelium. It contains large numbers of plasma cells and lymphocytes. Occasionally foreign-body giant cells are visible.

Mononuclear phagocytes containing pigment granules and others having an eosinophilic cytoplasm, suggestive of phagocytized lens cortex, are seen particularly adjacent to lens fragments.

The polymorphonuclear leukocytic response is moderate, and eosinophilic leukocytes are not evident.

The ciliary body is atrophic, its musculature small, its epithelium thinned, and its processes displaced anteriorly along the plane of the cyclitic membrane described above. Posteriorly the choroid is moderately compressed and is remarkably free from inflammation.

Ganglion cells are plentiful, and there is no evidence of inflammatory change in the retina. A scattering of mononuclear phagocytes and chronic inflammatory cells are seen in the anterior vitreous adjacent to the internal limiting membrane. There is some hyalinelike thickening of the walls of the smaller branches of the central retinal arteriole.

The optic nerve is not atrophic.

Opinion. Penetrating wound of cornea (surgical); secondary cataractous membrane; endophthalmitis phaco-anaphylactica.

Comment. There is no evidence of a chronic granulomatous uveitis. The extensive anterior synechia is suggestive of a leaky wound of long standing and a flat anterior chamber postoperatively. The chronic inflammatory reaction in and around the anterior uvea, and the presence of giant cells and polymorphonuclear leukocytes, in relation to cataractous lens remnants, supports the diagnosis of reaction to lens protein.

CASE 4

July 30, 1948. A man, aged 42 years, suffered a perforating injury, right eye, resulting in a blind, irritable eye.

May 22, 1949. Left eye became inflamed.

May 22, 1949. Right eye removed.

Description of pathologic anatomy

The sclera and cornea are greatly thickened, suggesting early atrophy of the globe. Centrally, there is a healed penetrating wound of the cornea. The surrounding stroma is vascularized and contains nests of chronic inflammatory cells.

Posteriorly, the wound is adherent to the pupillary portions of each iris leaf, and an organized membrane, consisting of iris tissue, cataractous lens remnants, and chronic inflammatory tissue, divides the anterior chamber into two compartments.

The peripheral cornea is vascularized and somewhat thickened. Descemet's membrane is folded and wrinkled as it approaches the anterior synechia centrally. Small loosely adherent peripheral anterior synechias occlude the trabecular meshwork on each side.

The iris is atrophic, its pigment epithelium thinned. It contains lymphocytes, plasma cells, and "clump cells," diffusely scattered through its stroma. Centrally, near the pupillary membrane, the inflammatory reaction is more pronounced. Nests of lymphocytes and plasma cells are seen destroying the normal architecture of the stroma.

Cataractous lens remnants are bound to the posterior surface of the iris and to the pupillary membrane. Lymphocytes, plasma cells, and giant cells of both the foreign-body and Langhan's type surround the lens, binding it to the pupillary membrane anteriorly, and to a cyclitic membrane posteriorly.

Pale-staining phagocytes resembling epithelioid cells, but not occurring in tubercles, are noted within the cyclitic membrane.

There is marked proliferation of the nonpigmented epithelium of the ciliary body. Mononuclear phagocytes are evident, and occasional nests of polymorphonuclear leukocytes undergoing necrosis are seen within the lens cortex. Fragments of lens capsule and lens epithelium are present.

The retina is detached from the disc to the ora serrata. Lymphocytes are seen perivascularly. Anteriorly the retina is involved in the cyclitic membrane. Cholesterol crystals are seen within the subretinal fluid. There are also large numbers of pigment laden phagocytes.

A few lymphocytic foci are seen throughout the choroid, but there is surprisingly little inflammatory change in this structure.

Opinion. Penetrating wound of eyeball; traumatic detachment of retina; traumatic cataract; traumatic iridocyclitis; endophthalmitis phaco-anaphylactica.

Comment. The presence of giant cells, polymorphonuclear leukocytes, and phagocytes invading the lens cortex establishes the diagnosis of endophthalmitis phaco-anaphylactica. There is no evidence of chronic granulomatous uveitis.

CASE 5

February, 1949. A man, aged 58 years, suffered a perforating injury, right eye, folowed by a traumatic cataract and persistent uveitis.

April, 1949. The right eye was enucleated. No history relative to left eye was obtained.

Description of pathologic anatomy

An intraocular foreign body, 3.0 by 3.0 mm., was removed from the temporal side of the eye.

Microscopically, the corneal epithelium is swollen and edematous. It is irregular in outline and thickness and contains numerous intra-epithelial bullae. Lymphocytes and plasma cells are seen densely packed in the perilimbal connective tissue.

Centrally, there is a healed penetrating wound, the posterior surface of which attaches to each iris leaf by an organized inflammatory membrane. The ruptured ends of Descemet's membrane are seen at each edge of this wound posteriorly.

Blood vessels and acute and chronic inflammatory cells are seen within the corneal stroma and are most concentrated within two mm. of the wound. Peripherally, in the stroma there are areas showing paucity of cells suggesting an acellular necrosis. Blood vessels are seen within the midstroma peripherally.

The temporal insertion of the ciliary body and origin of the iris root have been partially dialyzed. There are small, loosely adherent peripheral anterior synechias occluding the trabecular meshwork. This meshwork is sclerosed, and it and the anterior chamber contain lymphocytes, plasma cells, and mononuclear phagocytes.

The iris shows many breaks in its pigment epithelium. The stroma is thinned, and is densely infiltrated with plasma cells and lymphocytes. "Clump cells" are seen within the stroma, more in the pupillary portion.

Fig. 4 (Irvine and Irvine). *Case 5.* Polymorphonuclear leukocytes invading the lens.

A central inflammatory membrane occludes and secludes the pupil, binding the iris leaves and central cornea anteriorly, and is attached to an organized inflammatory membrane and cataractous lens remnants posteriorly.

The lens epithelium is seen as a single layer at the equator nasally, but it is otherwise not apparent. The lens capsule is ruptured anteriorly. Its cortex shows marked globule formation and is densely infiltrated with polymorphonuclear leukocytes (fig. 4) and cortex-laden phagocytes. These cells, undergoing necrosis and organization, are seen anteriorly between the lens and the iris leaves.

Equatorially on each side, large numbers of eosinophilic polymorphonuclear leukocytes are noted. Giant cells are rare. The lens capsule is not apparent posteriorly and temporally, and here there is also invasion of the cortex by acute inflammatory cells and mononuclear phagocytes.

The vitreous cavity contains a small amount of eosinophilic amorphous material. Plasma cells, occasionally lymphocytes, and moderately large numbers of mononuclear phagocytes with eosinophilic cytoplasm are seen. The last are more prominent anteriorly, and just behind the ora serrata nasally are seen in large clumps upon the internal limiting membrane.

The retina is edematous and vacuolated. Its nerve-fiber layer is infiltrated with lymphocytes and

Fig. 5 (Irvine and Irvine). *Case 6.* Eosinophils in exudate adjacent to lens material.

plasma cells that tend to accumulate about the walls of blood vessels. Similar changes are seen in the optic-nerve cup. Ganglion cells are plentiful. There are focal areas where the nuclear layers tend to merge and the normal retinal layers lose their continuity.

The inflammation in the choroid is minimal. Nests of lymphocytes are seen anteriorly but no tubercles of epithelioid cells to indicate chronic granulomatous inflammation. There is little atrophy of the optic nerve.

Opinion. Penetrating wound of eyeball; intraocular foreign body; traumatic cataract; traumatic endophthalmitis; endophthalmitis phaco-anaphylactica.

Comment. Mature cataract, polymorphonuclear, neutrophilic, and eosinophilic leukocytes in and about the lens and mononuclear phagocytes taking up lens cortex, plasma cell infiltration of the anterior uvea, and occasional giant cells establish the diagnosis of endophthalmitis phaco-anaphylactica.

CASE 6

June 20, 1948. The left eye of a man, aged 51 years, sustained corneal laceration which was repaired surgically.

June 30, 1948. Cataract extraction and iridectomy were performed. The eye became painful, blind, and showed evidence of shrinking.

August 10, 1948. Left eye enucleated.

Description of pathologic anatomy

The corneal epithelium is edematous and contains bullae. Two healed penetrating wounds are seen, one about two mm. from the nasal limbus, the other at the midportion of the cornea. A peripheral anterior synechia, about four mm. in length, obliterates the angle and occludes the scar seen nasally.

The central wound attaches to an organized inflammatory membrane posteriorly, thus dividing the anterior chamber into two separate compartments. The chamber angle is open temporally. Descemet's membrane is somewhat thickened at its periphery, and the endothelium is flattened and attenuated throughout.

The anterior chamber contains a moderate amount of amorphous, eosinophilic, fibrinous material, and a moderate number of plasma cells, lymphocytes, eosinophilic leukocytes, and mononuclear macrophages are seen along the endothelium, in the angle, and along the anterior surface of the iris and the pupillary membrane.

The mononuclear phagocytes tend to agglutinate and form clumps. They have distinctly straining chromatin material and easily visible nucleoli. Some have pale-staining cytoplasm; others contain granules of phagocytized pigment.

The pupillary portion of the iris leaf nasally is absent. Its ciliary portion and the iris leaf opposite are fused by an organized inflammatory membrane consisting of cataractous lens remnants, lymphocytes, plasma cells, mononuclear phagocytes, and eosinophilic leukocytes.

These cells are seen scattered diffusely and focally through the iris stroma. Their concentration is greatest at the pupillary portion.

There is marked atrophy of the pigment epithelium, particularly in its pupillary and midpupillary portion. A pupillary membrane continues posteriorly behind each iris leaf where it consists primarily of cataractous lens remnants.

Lens epithelium, capsule, and bladder cells are evident. Large numbers of mononuclear phagocytes are seen around the lens fragments, and large numbers of eosinophilic leukocytes (fig. 5) are seen between these fragments and the posterior iris.

A chronic inflammatory reaction of less severity is seen in the ciliary body. Phagocytes are seen in the circumlental space and proximity of the ciliary processes and along the pars plana in the anterior vitreous.

Occasionally these phagocytes are seen posteriorly along the internal limiting membrane of the retina. There is some edema of the ganglion cell layer posteriorly. Ganglion cells are plentiful. Lymphocytes are seen around the vessels in the nerve-fiber layer as it enters the optic disc. There is little optic-nerve atrophy.

The choroid is not remarkable. There is no evidence of chronic granulomatous uveitis in the choroid or ciliary body.

Opinion. Penetrating wound of anterior eyeball; traumatic cataract; iridocyclitis, traumatic; lens-induced uveitis.

Comment. Giemsa-stained sections confirm the

overwhelming presence of eosinophils and eosinophilic granules in the pupillary membrane and the iris stroma. The mononuclear phagocytes, with nuclei resembling those of epithelioid cells, forming clumps in the iris and in the anterior chamber are noteworthy. The neutrophilic-polymorphonuclear and giant-cell response is minimal, and consequently the pathology in this case is not typical of endophthalmitis phaco-anaphylactica.

Because of the immaturity of the lens fragments seen, the case is included in Group I, although the pathology includes many of the characteristics of Group II.

CASE 7

The history in this case, that of a man, aged 48 years, is not available.

Description of pathologic anatomy

Microscopically, there is a small amount of perilimbal round-cell infiltration. Blood vessels and a few chronic inflammatory cells infiltrate the corneal stroma peripherally.

The anterior chamber contains serum and free blood. The angle is opened temporally. Nasally there is a complete dialysis of the ciliary body. Free blood is seen throughout the suprachoroidal space anteriorly and nasally.

The iris is heavily pigmented and a few phagocitized pigment granules are seen lining, and within, the trabecular meshwork. There is atrophy of the pupillary portion of each iris leaf. Pigment granules are seen in the pupillary space. There is surprisingly little inflammatory reaction throughout the iris and ciliary body.

The lens capsule is folded and wrinkled. The epithelium appears to be intact. The epithelium extends two mm. posterior to the equator. The cortex, represented as an eosinophilic amorphous mass containing clefts and globules, is densely invaded posteriorly by lymphocytes, plasma cells, a few necrotic polymorphonuclear leukocytes, and mononuclear phagocytes, their cytoplasm swollen with eosinophilic lens material.

This invasion is seen along the posterior surface of the lens, and includes the posterior third of the cortex primarily. There is little reaction at the equator, centrally, and practically none anteriorly.

A newly formed connective tissue extends across the surface of the ciliary body posterior to the lens. There is marked proliferation of the nonpigmented ciliary epithelium. Lymphocytes and plasma cells, pigment laden phagocytes, and connective-tissue elements are seen within this membrane. It attaches posteriorly to the retina which is detached from the disc to the ora serrata, and forms a folded funnel-shaped membrane.

Posteriorly, the optic nerve is drawn toward the interior of the globe. Large aggregates of lymphocytes and chronic inflammatory cells are seen around vessels within the retina, and focally throughout its substance. This inflammatory reaction is most dense anteriorly at the junction of the separated retina and the cyclitic membrane. There is free blood and serum beneath the retina. Phagocytes are seen in clumps and singly throughout this fluid.

The choroid and suprachoroid are distended with serum. There is surprisingly little evidence of inflammation in the posterior segment. There is no evidence of chronic granulomatous uveitis.

Opinion. Penetrating wound of anterior eyeball; intraocular hemorrhage; traumatic separation of retina; traumatic cataract; phacogenetic endophthalmitis.

Comment. Although the absence of giant cells and the minimal polymorphonuclear response are not consistent with the diagnosis of endophthalmitis phaco-anaphylactica, nevertheless, the heavy invasion of the lens with plasma cells and lymphocytes suggests reaction to lens material. Therefore, this case could be put in Group II. It is included in Group I because of the immaturity of the cataract.

CASE 8

1946. A woman, aged 64 years, had a cataract extraction of the left eye.

1947. A cataract extraction of the right eye was performed.

1948. Glaucoma developed in both eyes, complicated by a chronic uveitis in right eye.

June 20, 1949. Pain in the right eye and secondary uveitis in the left eye aroused suspicion of sympathetic ophthalmia and the right eye was removed.

Description of pathologic anatomy

There is a healed penetrating wound one mm. inside the limbus above. Corneal epithelium is elevated above Bowman's membrane and separated from it by free blood and newly formed connective tissue for two mm. Elsewhere the epithelium is irregular in thickness, and in some places reduced to two to three cell layers.

Inferiorly the stroma is vascularized in the middle third throughout two mm. of the cornea peripherally.

Above, in the area of the wound, the cornea is thickened, vascularized, and contains foci of lymphocytes, plasma cells, and young fibroblasts. The perilimbal corneal tissue is densely infiltrated with round cells. The posterior surface of the corneal wound forms a dense anterior synechia with the remaining ciliary portion of the iris root, above, and organized cataract lens remnants extending to pupillary portion of iris root below.

A few chronic inflammatory cells and mononuclear phagocytes are scattered along the endothelium. The synechia divides the anterior chamber into two parts.

There are very small peripheral anterior synechias occluding the posterior third of the trabecular meshwork. The latter is sclerosed and contains lymphocytes, plasma cells, and pigment granules. Amorphous eosinophilic debris is seen in the anterior chamber.

The iris and anterior portion of the ciliary body are diffusely infiltrated with lymphocytes and plasma cells, the latter predominating. A small amount of free blood is seen within the stroma of the iris root below. Inflammatory reaction is more pro-

nounced in the pupillary irises and throughout the dense pupillary membrane. Here giant cells of both foreign-body and Langhan's type are seen.

Lens epithelium and bladder cells comprise the cataractous remnants. A few pigment-laden phagocytes are seen between the iris leaf and lens capsule. Polymorphonuclear leukocytes are not seen. There are occasional nests of mononuclear phagocytes invading the lens cortex above.

Ciliary processes are thinned and atrophic. Free blood is seen in the anterior vitreous along the pars plana. A large focus of mononuclear phagocytes having eosinophilic cytoplasm is seen along the internal limiting membrane at the ora serrata above.

There is some cellular hyperplasia of the media of the retinal arterioles. Perivascular lymphocytic infiltration is seen throughout the nerve-fiber layer.

Ganglion cells are present and may be reduced in number but this is difficult to judge because of the plane of the section. There is little gliosis of the nerve-fiber layer at the nervehead, and no posterior displacement of the lamina cribrosa.

Chronic inflammatory cells are seen around the vessels in the optic cup.

Choroid and sclera, aside from being somewhat thin posteriorly, are not remarkable. There are no aggregates of epitheloid cells in the uveal tract such as are essential for a diagnosis of granulomatous uveitis.

Opinion. Penetrating wound (surgical); incarceration of iris and lens remnants in wound; traumatic iridocyclitis; lens-induced uveitis.

Comment. The absence of polymorphonuclear cells invading the lens is against the diagnosis of endophthalmitis phaco-anaphylactica. The invasion of the lens with mononuclear cells, and the presence of giant cells, indicates reaction to lens material, and this case is placed in Group I because of the presence of immature cataractous elements.

CASE 20

March, 1948. A woman, aged 71 years, had a cataract extraction, left eye. Postoperatively, the patient became psychotic and the eye was injured. Uveitis ensued.

July, 1948. The right eye became inflamed, hard, and painful. Vision was reduced to light perception.

March, 1949. The right eye (sympathizing eye) was enucleated because of pain.

Description of pathologic anatomy

Microscopically, the uveal tract is densely infiltrated with lymphocytes and plasma cells. Nests of pale-staining epithelioid cells are seen. These are more frequently encountered anteriorly in the iris, ciliary body, and choroid behind the ora serrata.

Occasional giant cells are seen. These are of the foreign-body type and are seen behind the iris. The choriocapillaris is spared. Occasional proliferations of the pigment epithelium, resembling Dalen-Fuchs nodules, are seen.

There is perivascular lymphocytic infiltration throughout the retina. Macrophages are seen scattered throughout the vitreous anteriorly and posteriorly. The normal architecture of the ciliary body and iris has been destroyed by inflammation.

Cataractous lens remnants enmeshed in an organizing polymorphonuclear leukocytic exudate are seen behind the iris. Serum and exudate fill the anterior chamber. There is marked, chronic inflammatory infiltration of the perilimbus. The cornea is diffusely edematous.

Opinion. Chronic granulomatous uveitis; sympathetic ophthalmia; lens-induced uveitis.

Comment. Well-formed tubercles are not seen, and the giant cells could be explained on the basis of lens reaction. However, because of the sparing of the choriocapillaris and the destruction of the iris and the suspicious areas of pale-staining cells, this is lens-induced uveitis with coincident sympathetic ophthalmia.

(To be continued)

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A NEW VASODILATOR IN OPHTHALMOLOGY*

JOHN J. STERN, M.D.
Utica, New York

Priscoline is a relatively new vasodilator which has received considerable attention in Europe but has been introduced into this country only recently. It is 2-benzyl-imidazoline hydrochloride, one of a new group of compounds containing the imidazoline ring, many of which affect the circulatory system. Priscoline is the most potent vasodilator of them. It can be used subcutaneously, intravenously, intramuscularly, and by mouth. In ophthalmology, it can be administered by subconjunctival and retrobulbar injections.

Hoang-Xuan and Bailliart[1] treated 16 cases of chronic glaucoma with retrobulbar injections (1.0 cc of the 1.0-percent solution) once a week, or more frequently up to once daily, and tried to prolong the effect with oral or intramuscular administration.

Improvement of the fields was only slight and not constant, but visual acuity improved, and permanently so, in most cases. In some instances, the intraocular pressure decreased after four or five days. The authors observe that, even though the vascular theory of the etiology of chronic glaucoma is still a matter of discussion, vasodilation improves visual acuity in a great number of cases.

Bailliart[2] followed up this paper with a systematic study on the effect of priscoline on the capillary vessels of the retina. Retrobulbar injection of 10 mg. resulted in marked dilation of the peripheral arterioles and the perimacular vessels. An increase of speed of the erythrocytes in the retinal capillaries was observed, but no dilatation of the central vessels of the retina was seen.

This statement does not, however, agree with Roessler's[3] observation that instillation of a 10-percent solution of priscoline into the conjunctival sac was followed by a dilatation of the arterial diameter up to 89 percent.

Bailliart also failed to find a lowering of the arterial blood pressure in the eye after priscoline injections, while Fanta[4] had previously reported a fall of about 15 mm. Hg after intravenous administration. Bailliart assumes that functional disturbances of the retina are generally based on a circulatory disturbance, especially of the capillaries.

Retinal edema, small hemorrhages, and degenerative signs are the common outcome of this. Priscoline proved its therapeutic value in 16 patients, belonging to this group, suffering from macular edema, capillarosis, and senile degeneration of the macula.

Bendel[5] found priscoline to be effective in several cases of occlusion of the central retinal artery, and Simonelli[6] made the same observation in those cases where a spastic occlusion was present. Organic occlusions responded poorly or not at all. The latter author also observed a favorable course of optic neuritis with the use of priscoline, and very good results were achieved in keratitis and burns or corrosions of the cornea when drops, ointment, or subconjunctival injections were used.

Fanta[7] reaffirmed that retrobulbar injection of priscoline results in a fall of the intraocular pressure and reported good results in burns, glaucoma, and angiospastic conditions.

The combination of priscoline, which lowers the blood pressure in the vessels of the eye, and amylnitrite, which raises the general blood pressure, was considered ideal in embolism of the retinal arteries. Retrobulbar neuritis and alcohol-tobacco amblyopia responded favorably and, in 14 cases of methyl alcohol poisoning, improvement was observed if retrobulbar injections were given early enough.

* From the St. Lukes-Memorial Hospital Center, Memorial Division.

Jaensch and Budde[8] found that the retrobulbar injection of priscoline was very effective in circulatory disturbances of the retina and the optic nerve, particularly when pathologic alterations were still developing. Cases of hypertensive retinopathy with hemorrhages, intoxication amblyopia, and retrobulbar neuritis benefited greatly if no irreparable organic changes had taken place.

Case reports

In the following cases of different eye conditions, priscoline was used only in the form of 25-mg. tablets, three or four times daily. The side effects (formication, hot flushes, chilliness) were never embarrassing, and the nausea, which is encountered occasionally because of the effect of the drug on the gastric mucosa, could be controlled with antacids. The results of this treatment in some of the cases were encouraging and seem to deserve publication.

Case 1

F. D., a white woman, aged 53 years, was first seen in May, 1949, for correction of her presbyopia. Corrected vision in both eyes was 20/20, and the fundi showed mild hypertensive retinopathy with increased light reflexes and some arteriovenous nicking of first degree. In July, 1950, she returned with complaints of reduced vision in the left eye.

Vision in this eye was 20/70 corrected. In addition to the fundus changes seen previously, numerous discrete yellowish spots could be seen in the macula. A diagnosis was made of capillarosis and the patient was instructed to consult her family physician for a general check-up.

This examination was essentially negative except for a mild essential hypertension and, when she returned three days later, vision had further deteriorated to 20/400. The macular region showed some edema and the previously seen yellowish spots. Priscoline was prescribed (four times daily, one tablet), and three weeks later vision was 20/60. The retinal changes in the left eye seemed unaltered but the edema was less pronounced.

Five weeks after the onset of the symptoms, vision was again 20/20, the spots in the retina were hardly discernible, and the patient has remained well up to the date of writing.

Case 2

A. C., a white man, aged 48 years, complained of the sudden appearance of rainbow colors in the left eye four days prior to the first examination. He described a central positive scotoma with ragged edges which had been improving.

Vision in the left eye was 20/40; tension was 25 mm. Hg (Schiøtz) in both eyes.

The left fundus showed some tortuosity of the veins, there was some grayish retinal edema close to the upper temporal edge of the disc, and three small flame-shaped hemorrhages could be seen in this region close to the vein trunks.

The tangent screen field showed a large relative arcuate scotoma, originating from the blindspot and running underneath the fixation point to a point 15 degrees from it temporally (fig. 1).

A physical check-up failed to reveal any pathologic condition except hypertrophic, cryptic tonsils. Blood pressure was 130/70 mm. Hg. The diagnosis of early juxtapapillary choroiditis was made.

The patient refused hospitalization and was started on four tablets of priscoline daily. Five days later there was a slight improvement in the fundus picture; nine days later vision had improved to 20/30, the hemorrhages and the venous stasis had disappeared, and the scotoma could no longer be elicited. The patient has remained well up to date (eight months).

Case 3

A white man, aged 69 years, had noticed two weeks before his first visit that he could not see well in his left eye. The disturbance had cleared up somewhat and, when he was examined, vision was 20/30 in both eyes with correction. He stated, however, that on several occasions in the past few days he had experienced a "blacking-out" of the lower field of the left eye. This lasted for up to one hour after which it disappeared slowly, but returned, sometimes three or four times during one day.

The fundus showed hypertensive retinopathy with constricted arteries and arteriovenous nicking of second and third degree. There was a grayish,

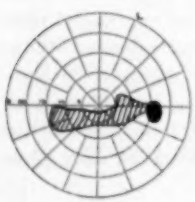

Fig. 1 (Stern). Visual field in Case 2 (August 14, 1950) for 10/1,000 red.

Fig. 2 (Stern). Visual field in Case 3 (July 31, 1950) for 10/1,000 white.

edematous area around a tertiary branch of the upper nasal retinal artery, about one-third disc diameter in size, partly obscuring the vessel. The field showed an absolute arcuate scotoma, starting about two disc diameters from the blindspot (fig. 2).

A general check-up revealed a blood pressure of 212/100 mm. Hg but no other significant signs. The patient was started on four tablets of priscoline daily, and four days later he reported that the attacks of altitudinal hemianopia were much less frequent.

Ten days after the first examination the arcuate scotoma could no longer be found, and no further "black-outs" had occurred. The focus in the retina was still visible but edema was much less marked and the artery now showed a continuous blood column.

Case 4

A white man, aged 33 years, had suffered from an optic neuritis in the left eye five years ago. In spite of several thorough check-ups in large hospitals, no cause for it had been found. It healed with a residual relative arcuate scotoma running from the blindspot to the point of fixation but not involving the latter. When the patient was first seen, he described a pulsating black spot in front of the right eye.

Visual acuity (with correction) was 20/20 in both eyes. The right disc was hyperemic and showed hazy borders without any real edema. The blindspot was slightly enlarged when charted under reduced illumination.

A physical examination revealed an acute rhinitis with a right maxillary sinusitis. The patient did not tolerate sulfa drugs or penicillin, so terramycin and local treatment for the sinusitis was initiated by an ear, throat, and nose specialist. At the same time the patient was started on priscoline.

The initial results were good and the positive scotoma decreased in size. However, the patient did not tolerate the drug which made him severely nauseated. Sodium bicarbonate took care of this complication, and the aspect of the nervehead became normal within three weeks. No enlargement of the blindspot could be found at that time. A check-up several months later was completely negative.

Case 5

A white woman, aged 64 years, came with a histor of increasingly poor vision in the right eye for the past two years. At the time of examination, she could only discern light and dark in the right eye. The left eye had also given her marked discomfort for the past few months, and she had received treatment in the form of eye ointments and vitamin A elsewhere. She was now complaining of a foreign-body sensation, redness, lacrimation and attacks of marked stabbing pains in the left eye and temple region.

Vision in the right eye was perception of light and dark; in the left eye, 20/40. The right cornea showed an almost complete opacity in all layers, with marked irregularity of the epithelium, which looked dry and exfoliating. The left eye showed a similar process, but it was only present in the lower half, just reaching but not covering the pupillary area. The left eye showed circumcorneal injection but no corneal vascularity. It was very irritable.

The corneal sensitivity in both eyes was greatly diminished, and the patient was unable to distinguish between the head and point of a pin over the periorbital and frontal regions. In the absence of any useful information in the history and the numerous preceding medical examinations, a diagnosis of neuroparalytic keratitis of unknown origin was made.

Priscoline was prescribed with a vague notion that vasodilation in the Gasserian ganglion or the supranuclear tracts might be of some benefit. The result was highly gratifying. The patient, who lived in another town, reported by letter that within a short period of time the very annoying neuralgic pain in the left side of the head had completely disappeared, that the eye was less irritated and that vision seemed to improve.

She was seen again two months after the first visit. Vision had improved to 20/30 in the left eye, the surface of the cornea in both eyes looked much smoother with no exfoliation present. The left eye was quiescent and white, but corneal sensitivity was still impaired. The process had certainly not progressed, and the previously opaque lower half of the cornea was clearer so that the iris could be discerned behind it.

Nine months after the initial visit, and two months after priscoline had been discontinued, vision was 20/25 in the left eye. Even in the right eye the opacities seemed to have cleared up somewhat, and vision had improved to counting of fingers at one meter. The eyes were quiescent, no discomfort was present, and the neuralgic pain had never bothered the patient again. Sensitivity of cornea and skin of the face was restored to normal.

Discussion

The diagnosis of neuroparalytic keratitis in Case 5 might be open to criticism in the absence of a traumatic lesion to the fifth nerve or any suggestion of a mechanical involvement of this nerve, like pressure from a tumor.

The disease has been observed in association with vascular lesions in the pons and in disseminated sclerosis but, in Case 5, it seems to have been an isolated process without any other systemic or local pathology. It

was, without doubt, a progressive process, and there is equally no doubt that the process was halted and even partly reversed by the use of a vasodilator.

The other cases herein presented are few in number and obscure as to their etiology. They seem to have a common angiospastic component, either in the arterioles or the larger vessels. Although the etiology might be different, vasodilator therapy was effective.

Two cases of occlusion of the central retinal artery, which were treated with priscoline tablets and amylnitrite, are not included in this report because they did not respond. Several other cases of senile degenerative processes in the retina have also been omitted because of the negative results obtained. They are mentioned here in order to prevent the impression that vasodilator therapy is to be regarded as a panacea.

That vasodilators have a definite place in ocular therapy cannot be doubted since Duggan[9] published his work on the effect of sodium nitrate in a number of eye conditions which he showed to be based on angiospastic processes. It would be attractive to speculate on the difference of the pharmacological action of the various vasodilators, but this aspect is intentionally avoided here. Nicotinic acid, amylnitrite, acetylcholine, sodium nitrite, priscoline, and so forth could probably be combined with advantage in cases where vascular spasm is suspected.

Summary

Vasodilator therapy in the form of oral administration of priscoline tablets was used in three cases of angiospastic processes in the retina, one of optic neuritis, and one of neuroparalytic keratitis. The satisfactory results recommend a trial with this preparation whenever angiospasm is considered to be responsible for similar processes.

3 Hopper Street.

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Ophthalmic Miniature

To find the place of entrance of the optic nerve, I fix two candles at ten inches distance, retire 16 feet and direct my eye to a point four feet to the right or left of the middle of the space between them: they are then lost in a confused spot of light; but any inclination of the eye brings one or the other of them into view. From the experiment here related, the distance of the centre of the optic nerve from the visual axis is found to be 16 hundredths of an inch; and the diameter of the most insensible part of the retina, one thirtieths of an inch.

Thomas Young, M.D., F.R.S.,
"On the Mechanism of the Eye,"
Transactions Philosophical Society, 1801.

THE RESPONSE OF THE RETINAL VESSELS TO PRISCOLINE IN VARIOUS VASCULAR CONDITIONS*

STUDY I

MORTIMER R. CHOLST, M.D., HAROLD F. SCHILBACK, M.D.,
MILTON B. HANDELSMAN, M.D., AND LEON M. LEVITT, M.D.
Brooklyn, New York

INTRODUCTION

Priscoline is a benzyl-imidazoline drug which, according to Grimson and his coworkers,[1] is an efficient general vasodilator. Pharmacologically, it has been shown[2] that there are two actions of the drug on the peripheral vascular system. In smaller doses it has a histaminelike action locally on the blood vessels and in larger doses it produces an adrenergic block. These changes have been studied chiefly in the extremities.

Europeon ophthalmologists have, however, used the vasodilatory property of priscoline clinically in many pathologic conditions, including central retinal artery spasm, macular degeneration, and glaucoma. The medication has been given orally, intravenously, retrobulbarly, and in conjunctival drops.

Guillamat, Hoang-Xuan, and Bailliart[3] employed priscoline injections in the treatment of senile and juvenile macular degeneration and reported improvement in the visual acuity. Hoang-Xuan and Bailliart[4] also treated patients with chronic glaucoma and found improvement in visual acuity but little change in the internal pressure or visual fields.

Physiologic studies of the effect of priscoline on retinal circulation have proven the vasodilatory properties of the drug, but the locus of the action has not been definitely established. The workers already mentioned (summarized by Bailliart[5]) report that, ophthalmoscopically, a dilatation of the central vessels of the retina could not be seen. No decrease in the blood pressure of the retinal vessels was found but a dilatation of the peripheral arterioles in the retina was observed.

In contrast to these observations, Roessler[6] has reported that ophthalmoscopic measurements of the central retinal arteries and veins demonstrate that instillation of a 10-percent priscoline solution in the conjunctival sac can lead to as high as 89-percent increase in the retinal arterial diameter.

Fanta,[7] also differing with the observation of Bailliart, found a decrease in the blood pressure in the retinal arteries after intravenous priscoline administration.

The observations of these last two workers were reported and confirmed by Miller[8] who found that the vasodilator effects of priscoline were particularly effective in the constricted retinal vessels of patients with hypertension. Using the Lobec method on the Gullstrand ophthalmoscope, he found that the drug acts as a dilator on spastic as well as normal arteries.

The difficulties inherent in measuring the width of the retinal vessels through an ophthalmoscope are well known. In order to help evaluate the effect of priscoline, it was decided to study its relative action on the retinal vessels in normal adults as well as patients with diabetes, nephritis, essential hypertension, and arteriosclerosis with fundus changes, by the method of angioscotometry.

Duke-Elder[9] defines angioscotometry as the tracing out of thin bands of physiologic blindness corresponding to the retinal blood vessels. According to Evans,[10] the normal angioscotoma corresponds to the particular retinal vessel arrangement as seen ophthalmoscopically. The widening phenomena coincide with known functional vascular vari-

* From the Department of Ophthalmology and the Department of Medicine, State University of New York College of Medicine, New York, and the Long Island College Hospital. The priscoline was supplied by the Ciba Pharmaceutical Company.

ations, and the form of widening follows the vascular distribution. Angioscotometry has been utilized to study the vascular action of many drugs such as histamine, caffeine, adrenalin, and nicotine.

Methods and Results

a. Technique

Various investigators have mapped out these shadows using different methods. The Evans[11] technique was followed and the stereocampimeter and charts were employed with a test object 0.4 mm. in size, spherical, and white. The illumination of the test chart was 15 foot-candles; monocular fixation was used.

The test object was moved from the seeing to the nonseeing area, and at all times at right angles to the border of the defect. The blindspot was first outlined and then the object was moved around the periphery of the blindspot until the superior arched angioscotoma was located and plotted. This study was limited to the changes that occurred to this band, since the time element, in these particular studies did not permit the mapping of the other bands which originate from the blindspot.

For purposes of classification in evaluating the response to priscoline in this study, the widening was classified as *good* if the band was widened maximally 4.0 degrees or more, *fair* if not widened more than 4.0 degrees maximally, and *poor* if not widened more than 1.5 degrees.

b. Procedure

The normal angioscotoma was plotted for each subject and then an intravenous injection of 50 mg. of priscoline was given over a one minute interval. The angioscotoma was then remapped approximately every five minutes. The time interval for maximum effect was about 15 to 20 minutes, following the injection. After its maximum effect the angioscotoma was recharted for variable lengths of time. The maximum dilatation was maintained for approximately 10 minutes to one-half hour, but several cases persisted for many hours.

c. Observations on healthy adults

As control cases, 12 normal individuals, eight male and four female, aged 23 to 42 years, with no previous history of vascular disease, were studied. A marked widening of

TABLE 1

Results of clinical study

Name	Age/Sex	Gen. Health	Angio. Widening	Funduscopy
Marked arteriosclerosis:				
1. A.T.	74/F	Marked A-S Chronic arthritis	Poor	*O.U.* Marked sclerosis of retinal vessels with irregularity of light reflex and increased tortuosity
2. W.T.	83/M	General arteriosclerosis	Poor	*O.U.* Marked sclerosis of retinal vessels with increased tortuosity and irregularity
3. A.S.	72/M	General arteriosclerosis Post second-degree burn of arm	Poor	*O.U.* Marked irregularity of light reflex and increased tortuosity of vessels. Venous compression at A-V junction
Mild arteriosclerosis:				
4. W.B.	51/M	Chronic arthritis	Good	*O.U.* Mild sclerosis of retinal vessels
5. W.K.	60/M	Postpneumonia	Fair	*O.U.* Moderate sclerosis with increased tortuosity of vessels

TABLE 1—(*continued*)

Name	Age/Sex	Gen. Health	Angio. Widening	Funduscopy
6. C.O.	55/M	Cancer of rectum	Good	*O.U.* Mild sclerosis of vessels *O.S.* Well-healed old chorioretinitis in periphery
7. A.H.	70/M	Chronic arthritis Postpneumonia	Fair	*O.U.* Moderate sclerosis and tortuosity of retinal vessels
8. G.D.	68/M	Pleural effusion (normal)	Fair	*O.U.* Moderate sclerosis of retinal vessels with irregularity of light reflex
9. G.P.	57/M	Low back pain	Good	*O.U.* Mild sclerosis of retinal vessels
Hypertensive heart disease:				
10. A.E.	58/F	Hypertensive H.D.	Good	*O.U.* Irregular caliber of arteries with increased light reflex
11. H.H.	65/M	Hypertensive H.D. Mild arteriosclerosis	Fair	*O.U.* Moderate tortuosity of veins. Nicking of veins at A-V junction. Irregularity of arteries
Essential hypertension:				
12. L.S.	38/M	Essential hypertension	Good	*O.U.* Increased light reflex in arteries. Increased tortuosity and nicking of veins at A-V junction *O.D.* One cotton-wool exudate near macula
13. A.W.	26/M	Essential hypertension (improved)	Good	*O.U.* Increased light reflex and change in calibre of vessels. Some irregularity in arteries and slight nicking of veins at A-V junction
Chronic glomerulonephritis:				
14. J.S.	33/M	Chronic glomerulonephritis	Good	*O.U.* Marked narrowing of arteries *O.D.* Two small hemorrhages superotemporal quadrant
15. H.W.	34/M	Chronic glomerulonephritis	Good	*O.U.* Arteries extremely narrowed. One flame-shaped hemorrhage near disc *O.S.* Two woolly exudates superior to macula
16. C.M.	40/M	Chronic glomerulonephritis	Fair	*O.U.* Arteries narrowed. Lipoid granules in periphery. *O.D.* Multiple flame-shaped hemorrhages near disc *O.S.* Several flame-shaped hemorrhages and three deep round hemorrhages between disc and macula
Diabetes without retinopathy:				
17. J.D.	25/M	Diabetes without retinopathy	Good	Negative
18. Y.F.	53/F	Diabetes without retinopathy	Good	Negative
19. L.H.	51/M	Diabetes without retinopathy	Good	Negative
20. A.S.	61/M	Diabetes without retinopathy Mild arteriosclerosis	Good	*O.U.* Mild irregularity of arteries

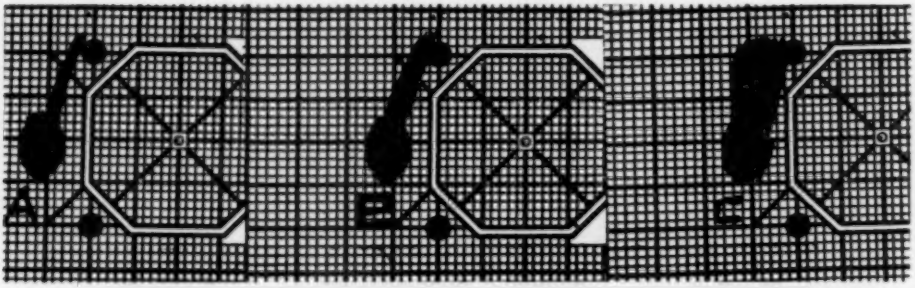

Fig. 1 (Cholst, Schilback, Handelsman, and Levitt). Angioscotoma plotted to show the effect of priscoline in a normal individual (R. C.). (A) Normal angioscotoma before priscoline. (B) Three minutes after the injection of priscoline. (C) Eighteen minutes after the injection of priscoline.

the angioscotoma was the response noted to priscoline in all of these individuals (fig. 1).

Eight of these cases were studied a second time with similar doses of normal saline instead of priscoline, without the patients' or examiner's awareness as to what medication was being used.

A maximum widening of the angioscotoma of one degree was noted in six individuals, and a maximum widening of one and one-half degrees was charted in the remaining two cases following injection with saline.

Six of these control patients were subsequently studied on three successive days, following a daily injection of similar doses of priscoline. All of these revealed only a slight difference in maximum widening of the angioscotoma each day it was mapped, with no change in the duration of widening. These individuals had not been informed as to what medication they were receiving each day.

d. Subjects

Twenty cases were studied at the Long Island College Hospital: two with hypertensive heart disease, two with essential hypertension, three with chronic glomerulonephritis, three with advanced arteriosclerosis, and four with diabetes without retinopathy. The remaining six patients ranged in age from 50 to 69 years, and these patients had arteriosclerotic changes—four moderate and two mild.

e. Results

The results of this clinical study (table 1) on 20 patients indicated poor response only in the three cases of severe arteriosclerosis. In five patients, only a fair response was obtained. Three of these cases were of moderate arteriosclerosis, while one patient had hypertensive heart disease and one patient had chronic glomerulonephritis.

Among 12 cases with good dilatation, three had moderate arteriosclerosis and were in the hospital recuperating from various intercurrent infections; two had essential hypertension; one hypertensive heart disease; two chronic glomerulonephritis and four diabetes without retinopathy (fig. 2).

Comment

From observations made during this study and findings in various vascular diseases (Elwyn[12]) it is believed that:

1. In cases in which there was visible evidence of marked vessel wall fibrosis, a definite nonresponse to priscoline was found. This was substantiated by the failure of the angioscotoma to widen. Because of the rigidity of these vessel walls together with a proportionate loss of elasticity, a vasodilatation could not have been anticipated and, on study, this was found to be true.

2. In the group of cases in which a moderate arteriosclerotic condition was seen, which we have classified as giving a fair response, a moderate widening of the angio-

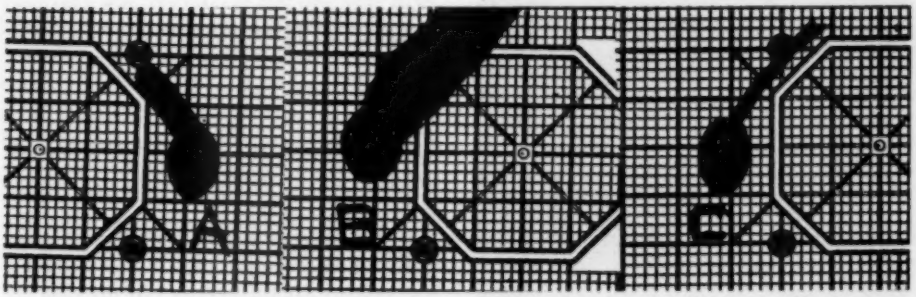

Fig. 2 (Cholst, Schilback, Handelsman, and Levitt). Angioscotoma plotted to show various responses to priscoline, 27 minutes after injection, in three different subjects. (A) Fair widening. (B) Good widening. (C) Poor widening.

scotoma was demonstrated. In this group of cases it would be anticipated that, because of the presence of a lesser degree of vascular pathologic process, some residual elasticity of the vessel wall remained. It is also known that, in this stage of vascular pathologic process, there is a relative degree of vessel-wall spasm.

3. The group which we have classified as good dilators responded by clinically evidencing marked widening of the angioscotoma.

In the third group—patients with vascular conditions accompanied by vessel spasm, with and without mild sclerotic changes—a good response to priscoline was noted. This appeared to confirm the findings in the first and second group where the sclerosis was relatively greater and spasm less.

Conclusion

1. In this study an established retinal vasodilator, priscoline, was utilized intravenously in dosage of 50 mg., thus employing its local vascular effect. Its relative effect on the fundus vessels in normal individuals, in diabetic patients without retinopathy, and in nephritis, hypertension, and various grades of arteriosclerosis with and without retinopathy, was studied by the method of angioscotometry. The maximum effect took place 15 to 20 minutes following injection.

2. The results would indicate that there is relatively a poor response to priscoline in visibly advanced retinal arteriosclerosis. However, in cases with moderate retinal arteriosclerosis, as well as other vascular conditions with various degrees of retinal spasm, a fair or good widening of the angioscotoma can still be obtained.

3. Normal individuals as well as patients with mild retinal arteriosclerotic changes revealed a good response to priscoline.

1 Nevins Street.

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IMPLANTATION CYST OF THE CONJUNCTIVA*

E. W. Perkins, M.D.
Richmond, Virginia

Implantation cysts of the conjunctiva are caused by trauma and are formed by the submucous inclusion of epithelium which proliferates. Implantation cysts, in general, arise by traumatic implantation of skin, epithelium, or hair bulbs into underlying connective tissue. Although common on the fingers and in the palm of the hand following puncture wounds, they are uncommon in the conjunctiva.

According to Parsons[1] the cyst increases in size by the secretion of the epithelial cells which are inverted, and probably by a desquamation and degeneration of the innermost cells. The contents of an implantation cyst vary according to the type of epithelium which produces it. It may contain clear watery fluid, mucoid material, hair, and sebaceous material. In the case of conjunctival cysts the contents are clear and mucoid in character.

A review of the literature reveals very little on the subject of implantation cyst of the conjunctiva. Ascott[2] reported a case following an operation for retinal detachment. The swelling was first noticed three months after operation in the bulbar conjunctiva near the lower fornix. It slowly increased in size and was removed nine months after the detachment operation. He believed the implantation of the conjunctival epithelium probably occurred while the conjunctival suture was being drawn from without inward. With this in mind he suggested the conjunctiva be held near the entrance of the suture, and kept on a stretch, to obviate this drag as much as possible.

Wolff[3] reported a case in 1928, the picture of which has been included in Duke-Elder's[4] textbook of ophthalmology. In his case the swelling began at the site of the wound produced by a piece of coal two years after the accident. Three years later the swelling had attained a considerable size, and protruded between the eyelids, preventing their closure. At operation the cyst was found to be between the conjunctiva and sclera. It contained clear, viscid, mucoid material and on section the thin wall was found to be lined with several layers of squamous epithelium.

Report of a case

A white man, aged 55 years, was seen at McGuire Veterans Hospital, Richmond, Virginia, in February, 1949, complaining of a painless swelling of the left eye which caused the upper lid to bulge.

He gave a history of the eye being hit two years previously by a flying splinter while chopping wood. However, this trauma had cleared without difficulty. Approximately nine months before coming to the hospital, he noticed a swelling and pressure under the upper lid. The swelling had gradually increased in size and had been painless.

Examination of the eye showed a bulging of the upper lid, which when retracted showed a subconjunctival cystic mass at the superior border of the limbus (fig. 1). The mass measured 12 by 10 by 6 mm. The examination of the eye was otherwise normal.

Operation was done under local anesthesia, and the cyst was found to lie between the conjunctiva and sclera. It was fairly loosely attached except at the base where it was firmly adhered to the underlying episcleral tissue. The cyst was removed intact (fig. 2).

* From the Ophthalmological Service, Veterans Administration Hospital, Richmond, Virginia. Published with the permission of the Chief Medical Director, Department of Medicine and Surgery, Veterans Administration, who assumes no responsibility for the opinions expressed or conclusions drawn by the author. Read before the joint meeting of the Virginia Society of Ophthalmology and Otolaryngology and the West Virginia Academy of Ophthalmology and Otolaryngology at White Sulphur Springs, West Virginia, May, 1950.

Recovery was uneventful and one year after operation there has been no recurrence.

Pathologic examination showed a thin-walled, uniloculated cyst, which contained clear mucoid fluid and collapsed when cut. Microscopic examination showed a cystic structure which was lined by flattened squamous epthelium which was supported by a thin layer of connective tissue (fig. 3). The diagnosis was epithelial implantation cyst of the conjunctiva.

Fig. 1 (Perkins). Implantation cyst of conjunctiva.

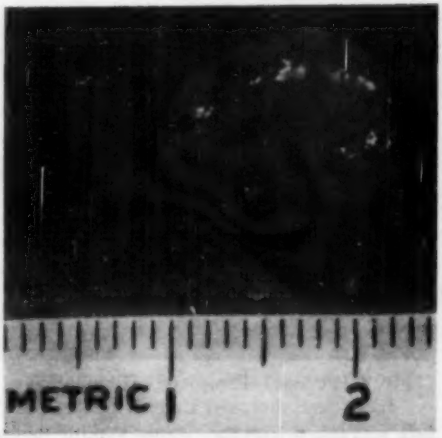

Fig. 2 (Perkins). Implantation cyst intact after removal.

Fig. 3 (Perkins). Microscopic section of wall of implantation cyst.

Similar conjunctival cysts

When making a differential diagnosis of conjunctival implantation cysts one must consider the somewhat similar types such as pseudocysts and the cystoid formations. The cystoid formations are found in operative scars as, for example, in trephinations for glaucoma. The pseudocysts are formed when the aqueous escapes under the conjunctiva in cases of ulcerative or traumatic perforation.

A patient with a so-called pseudocyst, which was similar in appearance to the conjunctival implantation cyst, was seen at the same Veterans Hospital in Richmond.

The case was that of a white man, aged 25 years, who had received a perforating injury to the eye when struck with a screw driver while overseas in 1944. Due to the eye injury he had been hospitalized for three months, but afterward had been returned to duty. Since the injury, there had been a small bleb at the limbus.

Two years after the injury he was seen at the Veterans Hospital and presented the picture which is shown in Figure 4. The pic-

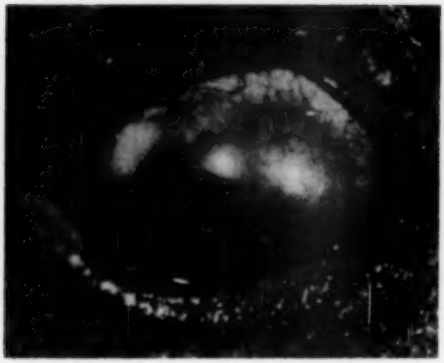

Fig. 4 (Perkins). Pseudocyst of conjunctiva.

Fig. 5 (Perkins). Eye six weeks after removal of pseudocyst.

ture was taken with the patient looking down slightly. The cystic glistening translucent swelling at the superior limbus measured 12 by 8 mm. Also, there was a linear corneal scar from the one-o'clock position to the center of the pupil with an anterior synechia and a tear in the iris sphincter. The tension was 35 mm. Hg (Schiøtz).

At operation the cyst was found to be a dissecting bleb with a fistula from the anterior chamber. When it was excised it seemed to be chiefly in the sclera, although the anterior wall was thin and easily ruptured. Following a keratome incision at the limbus at the 12-o'clock position, the anterior synechia was freed and a broad-base iridectomy was done (fig. 5). The patient was followed for three years and there was no recurrence of the cyst and the tension remained normal.

Summary

Implantation cysts of the conjunctiva are rarely encountered. A typical case of conjunctival implantation cyst is presented. A similar type of cyst, the so-called pseudocyst, which is also rare and which might be confused with an implantation cyst, is described.

509 Medical Arts Building (19).

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PSEUDOGLIOMA*

A clinicopathologic study of 15 cases: Part II. Pathologic classification

T. E. Sanders, M.D.
Saint Louis, Missouri

Pathologic classification

It has been accepted as obvious that the group of pseudogliomas consisted of several different types of pathologic lesions. It has even been quite generally agreed what these conditions were. However, the relative frequency, the exact nature, and the pathogenesis of these lesions is still not clear.

Reese[35] has said that retinoblastoma must be differentiated from the following conditions: (1) Retrolental fibroplasia; (2) metastatic retinitis; (3) massive retinal fibrosis; (4) Coats's disease; (5) diktyoma; (6) astrocytoma; (7) extensive medullated nerve fibers.

In the discussion on the differental diagnosis of retinoblastoma in his textbook, Duke-Elder[36] has said "that pseudoglioma consists of:

"1. Congenital abnormalities, especially those due to a persistence of the fibrovascular sheath of lens.

"2. Inflammatory conditions, such as organized inflammatory tissue in the vitreous, tuberculomata, gummata, exudative retinitis (of Coats), metastatic ophthalmitis, and so on, while in the earlier stages an exudative choroiditis may cause enxiety.

"3. A simple detached retina.

"4. Other neoplastic conditions, such as angiomatosis, and so forth.

"5. Cysticercus."

Wetzel[15] has stated that the causes of pseudoglioma from his survey of the literature are: (1) Metastatic ophthalmia from the acute infectious diseases of childhood; (2) tuberculosis or, much more rarely, congenital syphilis, giving rise to granulation tissue proliferation; (3) traumatic intraocular hemorrhage during birth or after birth. (4) detachment of retina caused by birth trauma or intrauterine inflammatory process; (5) congenital malformation and fetal remains; (6) Coats's exudative retinitis and von Hippel's angiomatosis retinae.

Although all of these conditions are hypothetically possible, several are not represented in this series or in the literature. I could find no reported case of pseudoglioma from astrocytoma, extensive medullated nerve fibers, or cysticercus. Astrocytoma is a very rare tumor of the retina, but it is a very unlikely cause of glioma as it is a condition of adults (McLean, 1937[37]).

Although detachment of the retina is often found in pseudoglioma, it is usually an inflammatory detachment with direct involvement of the retina. Simple detachment of the retina is a very rare condition in children.[38] Examples of all the conditions mentioned by Wetzel[15] are found in the present series.

From these detailed histologic examinations of these 15 cases, an attempt was made to classify these pseudotumors on the basis of the site, the nature, and the probable pathogenesis of the lesion causing the mass that led to the mistaken diagnosis of true intraocular neoplasm. On this basis these cases fall into seven general classifications:

Type I. Persistence and hyperplasia of the posterior tunica vasculosa lentis (Case 6).

Type II. Organization of a vitreous mass forming a cyclitic membrane (Cases 4 and 14).

Type III. Chorioretinitis (Cases 9 and 13).

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TYPE IV. Exudative retinitis (Cases 1, 2, 5, 7, 8, and 10).

TYPE V. Massive retinal fibrosis (Cases 3 and 15).

TYPE VI. Retrolental fibroplasia (Cases 11 and 12).

TYPE VII. Other tumors, as diktyoma and angiomatosis retinae (not represented in this series).

Each of these types deserves some discussion of its detailed nature and the relationship of the present cases to the disease process. Especially interesting is the relationship of the various types to each other; for instance, persistence and hyperplasia of the posterior tunica vasculosa lentis to retrolental fibroplasia; exudative retinitis to retrolental fibroplasia; and exudative retinitis and massive retinal fibrosis to so-called Coats's disease.

TYPE I. PERSISTENCE AND HYPERPLASIA OF THE POSTERIOR TUNICA VASCULOSA LENTIS

In this particular series this condition is represented by one case (Case 6) (fig.1).

This case seems to be quite typical as there is a dense retrolental fibrous mass which is well vascularized and closely in contact with the posterior capsule. There is a widely patent hyaloid vessel which apparently runs from the disc into this mass. The epithelium of the lens capsule is found posteriorly and there is also a defect in the posterior capsule of the lens in the area of the attachment of the hyaloid vessels.

Fig. 1 (Sanders). *Case 6.* Persistence of posterior tunica vasculosa lentis. Shallow maldeveloped anterior chamber. Cataract with retrolental sheath. Cupping of disc with patent hyaloid vessel.

In addition, this particular eye has multiple developmental defects, as there is maldevelopment of the iris angle, giving rise to congenital glaucoma. The combination of these two conditions has been reported by Pagenelli[20] and has been stated by Terry[7] to be quite characteristic.

This condition has long been recognized, having been first reported in 1851 by Howard[39] and fully described by Nettleship[40] in 1872. Treacher Collins[13] in his series of pseudogliomas stresses the place of this lesion as a cause of pseudoglioma. Since then the literature of this condition has been quite extensive.[16, 20–21, 26, 41–43]

Terry[3] states that persistent tunica vasculosa lentis is formed by growth of connective tissue in the interstices of the vascular network which normally is a network of vessels alone without mesoderm. Normally this atrophies when the formation of aqueous begins. The anterior tunica vasculosa is not involved as it has a separate blood supply and, if persistent, forms a pupillary membrane.

This condition has been of much interest recently because the original papers of Terry[1–7] attributed retrolental fibroplasia to this type of lesion. Reese and Payne[8] enlarged on this idea in their paper on persistence and hyperplasia of the primary vitreous. However, recently, Reese[9] has stated that this condition and retrolental fibroplasia are two different, but related, entities. The relationship between these two conditions will be discussed under retrolental fibroplasia.

TYPE II. ORGANIZATION OF VITREOUS MASS.

This type has been widely recognized as the commonest type of pseudoglioma.[14] It is also the best understood from the standpoint of its pathogenesis. Treacher Collins[13] recognized this as the most common cause of pseudoglioma in his series. He stressed the presence of meningitis and ear infections. He stated that he thought the spread

to the eye of meningitis could be by blood stream or by direct extension.

Many of these cases are associated with infectious diseases of infancy and childhood of which there may be a bacteremia. In this type a small, infected embolus lodges in the choroidal or the retinal circulation, forming a localized abscess. This ruptures into the vitreous forming an abscess there, usually anterior. This may remain unrecognized until it organizes into the cyclitic membrane. The presence of the cyclitic membrane causes the white appearance of a mass behind the eye. Shrinkage of the membrane can continue to cause a soft degenerated globe.

In 1911, Coats and Forbes[44] reported four cases of metastatic ophthalmitis of the pseudogliomatous type in which the meningococci were found intraocularly. They thought the infection was metastatic with the primary lesion in the retina in three cases and in the choroid in the other. They estimated the incidence of this lesion as about four to five percent of the cases of meningococcal meningitis. They concluded that pseudoglioma is a clinical entity and that the majority of cases were due to the meningococcus.

In meningitis, Percival[38] believes that more endophthalmitis from this source should be expected in children than in adults as there is a more direct route of spread. He has shown that if fluid is injected into the sheath around the optic nerve in children, this will penetrate directly into the choroid, while in adults it meets a very definite block, either at the optic foramen or at the blind end of the optic-nerve sheath, In the same report he mentions the case of a two-year-old child who was recovering from meningitis in which there was pseudoglioma in one eye and retrobulbar neuritis in the other.

In 200 consecutive cases of cerebrospinal meningitis Lewis (1936)[45] found endophthalmitis in 15 cases, or 7.5 percent. He did not believe that this lesion was characteristic, but is seen in this disease more often than in any other. Only in four cases of the 15 was enucleation necessary, and none of these was for pseudoglioma.

Fig. 2 (Sanders). *Case 14.* Organization of vitreous hemorrhage. Complete detachment of retina with retrolental fibrous mass containing old blood.

Case 4 represents a typical example of this lesion both clinically and pathologically. The history of long undiagnosed fever could easily have been low-grade meningitis or some other systemic blood stream infection. The pathologic findings of the broad band of connective tissue immediately behind the lens attached to the ciliary body is quite typical of the organization of a vitreous abscess.

Case 14 (fig. 2) represents a smiliar pathologic result with a different etiologic background. Rather than organization of vitreous abscess, this case probably represents the organization of a massive vitreous hemorrhage. Similar cases have been reported by Zeeman[22] and Lackman.[46] There is not only much old hemorrhage present in the area immediately behind the lens, but there is a massive connective tissue membrane in which there is much scattered pigment. The organization of the blood would probably account for the peculiar discoloration of the iris as noted in the clinical history. However, as in this particular case there is no history of trauma to the eye after birth, it is possible that the vitreous hemmorrhage occurred before or during birth.

It is surprising that only two cases out of the 15 were of this type, considering that in the past this was the most common type.

However, it would be expected that in the past few years the incidence of this type of endophthalmitis would be decreasing because of the improved treatment of the blood stream infections with chemotherapy and the antibiotics. This postinflammatory type of vitreous organization will probably become more rare, while the second type, the organization of hemorrhage, will probably not be particularly influenced by modern therapy.

Type III. Chorioretinitis

Uveitis of the endogenous type is extremely rare in infants and young children. This lesion represents the rarest type of pseudoglioma as reported in the literature. It is, therefore, a little unusual that there are two cases of this type (Cases 9 and 13) in the present series. As will be suggested later, it is possible that these cases represent a previously unrecognized type of pseudoglioma.

Treacher Collins[18] in his series reported a case of massive choroidal tuberculoma. Wetzel[15] suggests the possibility of this type of lesion, and stated that von Hippel[23] believed that childhood tuberculosis is more likely to give this type of reaction than the type seen in the adult.

There have been a few other cases reported.[47, 48] These were massive conglomerate tuberculomas of the choroid. Apparently this lesion is becoming much more rare as all of these cases were reported in the literature many years ago. This probably is not only due to the lessened general incidence of tuberculosis, but probably also due to changes in the prevalent types.

Fig. 3 (Sanders). *Case 13.* Chorioretinitis with inflammatory detachment. Axial detachment of retina with large cyst. Retrolental membrane with active inflammation in retinal mass. Active choroiditis in posterior pole.

These two cases (Cases 9 and 13) (fig. 3) are entirely different from massive tuberculous lesions of the choroid, but are very similar to each other. They represent a massive inflammatory detachment of the retina with a small primary lesion of active choroiditis in the posterior pole. One (Case 13) showed an active granulomatous lesion in the retina itself, while the other (Case 9) showed secondary fibrosis of the retina probably postinflammatory. It should be emphasized that both of these cases were bilateral and in both very poor vision had been present since birth. In one (Case 9) the mother had a severe cold during the first trimester of pregnancy.

In the last eight or 10 years a form of bilateral, granulomatous, central choroiditis has been described as a congenital lesion. This is due to toxoplasma, a protozoan organism known for many years to infect animals.

The first reported lesion in the human with this organism was described in the eye by Janku in 1923.[49] He reported a case of a child dying of internal hydrocephalus in which a hyperplastic inflammation of the retina was found at autopsy. Histologically a protozoa was found in the retina which he considered to be sporazoa, but most authors since agree that these are typical toxoplasmas. The first proved human case was that reported by Wolf, Cowen, and Paige, in 1939,[50] in which the organism was found by inoculation and culture.

The reservoir for this infection is probably in various types of animals, particularly rodents, but the exact vector is not known. There are only a few reports of this condition occurring outside of the United States. A

complete review of the subject was presented by Callahan, Russell, and Smith[51] in reporting eight fatal cases.

That this organism is a definite cause of encephalomyelitis in young children was definitely proved by Sabin in 1941.[52] Acute adult type of toxoplasmosis was also reported in 1941 by Pinkerton and Henderson.[53] This condition resembles very closely Rocky Mountain spotted fever. There are probably many latent cases in adults as it has been estimated that in the St. Louis area 2.7 percent of the general population show evidence of toxoplasmic infection.[51]

The encephalomyelitis is the major involvement in children, characterized by convulsions, internal hydrocephalus, and other neurologic findings. Cerebral calcification as shown by X ray is a very common finding.

Serologic studies, as developed by Sabin and Olitsky,[54] have proved very valuable in the diagnosis of this disease. They showed that the plasma of infected persons had a neutralizing effect on these organisms when introduced into animals.

Heidelman[55] found that the serologic tests were positive in 63 percent of 27 patients with congenital chorioretinitis. In the mothers of these cases, six of seven had positive findings. Johnson[56] found an identical incidence of positive tests in congenital choroiditis cases, 20 positive from a total of 32.

The ocular lesions of toxoplasmosis have been described in detail by Koch, Wolf, Cowen, and Paige in 1943.[57] They reported a series of six cases, five of which had histologic examinations of the posterior segment removed at autopsy. In the same year Vail, Strong, and Stephenson[58] also reported a series of six cases, five congenital and one acquired. However, none of these had histologic examination of the ocular lesion.

Heath and Zuelzer[59] have reported the disease in twins, one surviving with a central chorioretinitis, and the other with typical findings at autopsy. In most of these cases the infection is probably acquired in utero from an infected mother.

The ocular lesion is usually bilateral, with multiple areas of acute necrotizing chorioretinitis. In practically all cases the central, macular area is bilaterally involved. As usually seen, these lesions are represented by sharply defined areas of healed chorioretinitis with the remainder of the retina not unusual. In the past these have often been called macular colobomas.

Occasionally there may be secondary proliferation in the area of the lesion. The media are uninvolved, but at times some secondary optic atrophy occurs. Often there is microphthalmos or other congenital malformation, probably due to the intrauterine infection.

Up to the present time, no case of pseudoglioma due to toxoplasmic chorioretinitis has been reported. All the cases examined histologically in the literature have been autopsied specimens. Koch, Wolf, Cowen, and Paige[57] definitely state that pseudoglioma must always be considered in the differential diagnosis of toxoplasmosis. They say, "It is not unreasonable to assume that some cases in which the diagnosis would now be pseudoglioma may prove to be instances of toxoplasmic chorioretinitis."

Inasmuch as these two cases represent bilateral granulomatous choroiditis present at birth and involving chiefly the posterior pole with massive secondary inflammatory detachment, it is very possible that these cases may represent true toxoplasmic chorioretinitis. It is interesting that in one case (Case 9) the mother had a "severe cold" during the first trimester of pregnancy. However, no organisms were found on microscopic studies in either case.

It is unfortunate that the blood of these children and their mothers is not available at the present time for neutralization studies. It would also be very desirable to obtain skull films in regard to possible intercranial calcification. Further investigation of these two cases will probably be done in the future in an attempt to substantiate the diagnosis of toxoplasmic chorioretinitis.

Fig. 4 (Sanders). *Case 1.* Exudative retinitis. Complete detachment of retina with localized area of inflammation.

Type IV. Exudative retinitis

The six cases making up this group are pathologically characteristic and resemble not only each other very closely, but a number of other cases described in the literature.[17, 27, 34] These cases may be divided into four early cases (Cases 1, 5, 8, and 10) (fig. 4) and two late cases (Cases 2 and 7).

Histologic characteristics of the early cases are:

1. A diffuse edema of all the retina with subsequent bullous retinal detachment by exudation of subretinal fluid. On the surface of the retina in contact with this fluid, there are a number of large "bladder cells" derived from the pigment epithelium of the retina. At times varying numbers of similar cells are found in the retina possibly by invasion.

2. There is usually one localized area of retinal inflammation and degeneration usually involving chiefly the inner layers with edema and infiltration of albuminous fluid, moderate, diffuse infiltration of chronic inflammatory cells, and secondary degeneration of the retinal elements with only slight replacement gliosis.

3. Lack of any retrolental membrane is seen; the anterior vitreous usually remains quite clear.

4. There is lack of any lesion whatsoever in the uveal tract except for an infiltration of the iris stroma with an albuminous fluid similar to that seen in the retina.

5. Practically no, or at the most a very slight, hemorrhage is present in the retina, but usually some gross dilatation of the retinal vessels with some occasional perivascular infiltration of lymphocytes.

The late cases represent a complete detachment of the retina with the retina in folds and irregular masses immediately behind the lens. There is less intense edema of the retina with less cellular infiltration. There is usually a thin connective-tissue membrane on the surface of the retina which tends to form later between the retina and the posterior surface of the lens. Usually the area of focal inflammation of the retina is represented by a mass of gliosis and newly formed vessels in the retina itself. These findings represent an advanced stage of the changes described for the early lesion.

Although the term exudative retinitis is very appropriate for this condition, it is used with some misgivings because of its usual association with so-called Coats's disease. If the pathologic picture already described is compared with that originally described by Coats,[60] it is evident that the two conditions are distinct conditions.

In Coats's classic description,[60] "Forms of retinal disease with massive exudation," he divided the cases into: (1) Cases without gross vascular disease; (2) those with gross vascular disease; and (3) cases with large arteriovenous communication.

He stated the principal characteristics of exudative retinitis were: The presence in the fundus of large masses of white or yellow exudate, usually some hemorrhage, and cholesterin crystals; the occurrence in young people, most commonly the male sex, usually unilateral involvement; and the perfect health of the patient.

Coats reported the histologic findings in six cases and reviewed the findings in other cases in the literature. He concluded that the primary lesion was a hemorrhage, usually

into the outer reticular layer with possible subsequent subretinal hemorrhage. Secondarily there was an organization of the hemorrhage into a white mass.

The course is slow and usually associated with vascular changes which may be either a primary vascular change or be secondary to the organization of connective tissue. He believed that the primary hemorrhage may be due to various causes.

In reporting four additional cases in 1912 under the title "Retinitis exudiva,"[61] he believed that hemorrhage was still the fundamental lesion, but that there was secondary inflammation associated with this.

Duke-Elder[62] states "that today, it is obvious that Coats' disease is by no means a clinical entity, but embraces a considerable number of conditions. Such cases with massive exudation between the retina and choroid may be classified as follows:

"1. External Haemorrhagic Retinitis (of Coats), haemorrhagic cases in which the element of haemorrhage seems undoubtedly to be primary and the disease seems essentially vascular in origin.

"2. External Exudative Retinitis (of Coats), (Sero-fibrinous degenerative retinitis of Leber) wherein haemorrhagic extravasations are absent or minimal and the disease is toxic in origin and vascular in nature.

"3. Angiomatosis retinae (of von Hippel and Lindau); (Massive exudation with arterio-venous communication of Coats); a congenital disease of neoplastic angiomatous nature which in its later stages has exudative tendencies.

"4. Retinal degeneration with multiple miliary aneurysms (of Leber), a disease of unknown aetiology, perhaps toxic, perhaps degenerative and perhaps related to condition preceding.

"5. Cases due to disturbances in the choroidal circulation. The majority may be termed senile exudative choroiditis (sometimes called the senile form of Coats' disease). Here the subretinal exudation is derived from arteriosclerotic vessels in the choroid. . . . More rarely it occurs in younger people owing to some obstruction in the choroidal circulation such as thrombosis of the vortex veins.

"6. Traumatic cases occurring in newborn infants, in whom a haemorrhage occurs preferentially from the outer plexiform layer and bursts through to the subretinal space to form a typical picture of Coats' disease."

On the other hand, Elwyn[63] in a recent very comprehensive review of Coats's disease believed that it is a distinct pathologic as well as a clinical entity. He thinks that the fundamental pathologic element is a vascular malformation involving a definite vascular area of the retina, this corresponding to a telangiectasia forming in the small terminal vessels. However, he does not distinguish as separate entities the exudative retinitis of the externica hemorrhagica type and the serofibrinous type of Leber.

That this confusion is not due to Coats himself is made clear by the following statement which he made when reporting an additional case in 1911.[64] He says "this case is a typical example of the condition outlined by me as 'Retinal Disease with Massive Exudation.' This nomenclature was used to avoid possible confusion with any other disease to which the name 'exudative retinitis' may have been applied; but since that term does not seem to have been appropriated to any well-defined entity, it may perhaps be employed as a less cumbersome name to designate the symptom group now under consideration. In view of the probable pathology of the affection, the alternative name 'Retinitis hemorrhagica externica' might be suggested."

It seems obvious that Coats's disease is a clinical term for a particular ophthalmoscopic picture caused by several different pathologic lesions. This is similar to the concept of pseudoglioma presented here, this condition also being a clinical entity made up of various pathologic lesions. Similarly, I do not believe that the term, Coats's disease, should be used as a final pathologic diagnosis.

Of the six classes listed by Duke-Elder, our TYPE IV, exudative retinitis, as will be shown, is identical with the serofibrinous type of Leber. His sixth group of retinal hemorrhage in the newborn is our TYPE V, massive retinal fibrosis, which will be discussed later.

Lamb,[26] in 1938, reported eight cases of exudative retinitis from this laboratory, six early cases and two late cases. Both of the late cases and one of the early cases were in adults. In the remaining five, the eyes were removed for suspected gliomas. One of these (Case 3) was placed in the massive retinal fibrosis group. To these four cases (Cases 1, 2, 5, and 7) originally described by Lamb, two cases (Cases 8 and 10) have been added.

Lamb emphasized that "Anatomically, the primary and essential change in each of the eight eyes is an infiltration of the retina in whole or in part with albuminous and edematous fluids, leading to an exudate of the same fluids under the retina and a resulting complete or partial detachment of the retina. The two late cases are further distinguished by the presence of large amounts of connective tissue between the choroid and the retina or in the retina." The two late cases are not included in this report, but appear to be the external hemorrhagic type.

Leber[25] was the first to suggest that some factor other than hemorrhage was primary and suggested an embolic or toxic origin, giving it the name of serofibrinous degenerative retinitis. Leber's idea of the pathologic reaction is, to quote Lamb,[26] ". . . serofibrinous degenerative retinitis consists of one to several circumscribed foci of inflammation and necrosis in the retina with serofibrinous exudation into the tissue and on its surface. Hemorrhages were more or less frequent but not, as a rule, considerable. Especially characteristic was the proliferation of the retinal pigmented epithelium. These proliferated epithelial cells, after taking up fatty material, built bulky formations. Part of these epithelial cells, containing small granules of fat, wandered into the retina although the larger amount remained in the subretinal fluid. Later, extensive retinal detachment occurred through the exudation of serofibrinous fluid from the retina. Lastly, an encapsulation of the subretinal and retinal fatty detritus took place by means of the new formation of fibrous tissue. Part of the latter at least appeared to be derived from the pigmented epithelium."

Marshall and Michaelson[27] also give a very excellent description of this condition in reporting four cases of their own and one other from the literature. They believe that their cases are identical with those described by Leber as retinitis exudativa serofibrinosa. They think that the retinitis is probably embolic-toxic in origin and definitely not the result of a preëxisting hemorrhage. They conclude that there are definite clinical and pathologic differences between three different entities, retinitis exudativa-hemorrhagica (of Coats), retinitis exudativa senilis, and retinitis exudativa-serofibrinosa of Leber.

From both the literature and the findings in these six cases, we must conclude that the serofibrinous type of exudative retinitis of Leber is a distinct pathologic entity. Its place as one of the commonest causes of pseudoglioma must be emphasized and the condition is probably more common than is supposed.

Its exact nature is still not clear. The albuminous edema, the dilatation of the vessels, the absence of marked inflammation or hemorrhage suggest a metabolic vascular lesion with changes in permeability of the capillaries. The tendency for localization in the inner layers in the region of the capillary layers would support this view. The areas of intense reaction with inflammation could be areas of extreme vascular change with a type of infarction. As will be discussed later, these changes may be related to those seen in retrolental fibroplasia.

TYPE V. MASSIVE RETINAL FIBROSIS

The two cases (Cases 3 and 15) (fig. 5) representing this condition in the present

series are very similar to each other, but quite different from all the other cases, inasmuch as they represent localized retinal disease without massive detachment.

The pathology in each case consists of a localized area of edema, cellular infiltration, and marked retinal degeneration. There is in the area a flat detachment with no evidence of involvement of the underlying choroid. In each area there is localized connective-tissue proliferation, not only in the retina, but also on its surface represented by strands of fibroblasts extending out into the vitreous.

In one case there is some old hemorrhage immediately under the retina. In each case there is a second localized area of folding and distortion of the retina with one of the vitreous strands attached to the retina in an anterior position. The large, apparently primary lesion in each case is in the region of the posterior pole.

It is obvious that this is the sort of lesion that is usually called Coats's disease. Rados,[66] in 1921, reported a very similar case in a one-year-old girl as one of retinitis exudative externa. As already mentioned, Lamb[28] included Case 3 in his report on exudative retinitis. However, as stated previously, I believe that Coats's disease is not a pathologic entity and that a more exact pathologic diagnosis can usually be made.

Reese,[25] in 1933, described three cases of an almost exactly similar condition under the term "massive retinal fibrosis." In two of his cases, the eyes were removed because gliomas were suspected. He thought that there was possibility of tumor because of the apparent progression of the lesion, with changes in the appearance of the mass due to continued activity. He thought the fundamental lesion in each of these cases was a hemorrhage in the retina with secondary inflammation and organization causing the retinal folds by contraction. In each of his cases there was a history of preëxisting trauma with a positive Perl reaction for iris pigment of hemorrhage.

There is no history of trauma in either

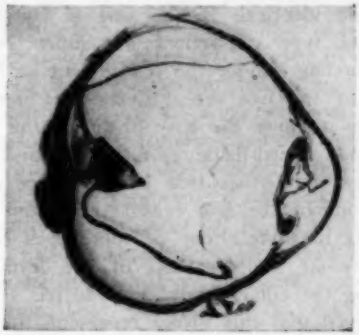

Fig. 5 (Sanders). *Case 15.* Massive retinal fibrosis. Posterior area in retina of inflammation and fibrosis. Anterior fold of retina with attached vitreous band.

of the present cases, and the Perl reaction was negative. In them the appearance of the strands in the vitreous look much like organization of hemorrhage of the retinitis proliferans type.

Klien,[67] in discussing retinitis proliferans, states there are two types in one of which the formation of connective tissue precedes formation of the blood vessels. There may be involvement of the retina following a hemorrhage into the vitreous or into the retina or following inflammation of the retina. The strands of connective tissue in the vitreous would appear to be of this type.

It is not certain in these particular cases whether the preëxisting condition was a hemorrhage or a localized retinal inflammation. Either of these could cause such fibrosis in a local area. These cases are so similar to those reported by Reese[25] that this condition is probably a pathologic entity for which Reese's term, "massive retinal fibrosis," is acceptable.

Type VI. Retrolental fibroplasia

There is very little pathologic material of this disease available, so there is no established picture of the exact pathology of this condition.

Reese and Payne[8] have described five such eyes in the advanced stage, these five being

almost identical. There was a complete funnel type of retinal detachment with hemorrhage under the detachment in four out of the five cases. Retrolental fibrous tissue occupies the base of the retina and extends varied distances into the funnel. In the retinal tissues true rosettes were seen in four of the five eyes and calcium deposits in three. Four of these eyes show embryonic malformation of the filtration angle, the ciliary body, and the iris. They believed that this group represents failure of formation of the secondary vitreous, so that there is no mass to hold the retina in place, causing the detachment. The later subretinal hemorrhage with organization and contracture also probably play a part in the detachment. In the membranes are highly vascularized areas not unlike hemangiomatous tissue. A number of their patients had hemangiomas of the skin, a tumor of increased incidence in prematures.

In a later paper Reese[9] believed that the pathologic process is centered around the base of the vitreous where there is much fibrous vascular tissue which probably undergoes some hyperplasia, but its increase seems to develop mostly from organization of hemorrhage. The resultant contraction leads to the detachment of the retina which seems invariably to be present. He states that the fibrous tissue extends over the posterior part of the lens, but for the most part posteriorly into the vitreous in the direction of the disc.

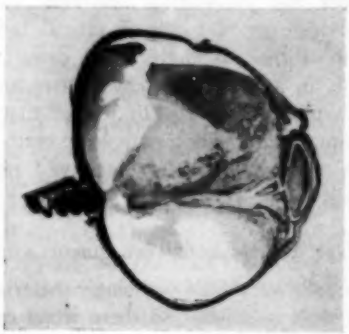

Fig. 6 (Sanders). *Case 11.* Retrolental fibroplasia. Massive retinal detachment with marked subretinal hemorrhage. Retrolental fibrous membrane. Shallow anterior chamber.

He believes the basic lesion of this condition is the persistence of some of the vascular primary vitreous in the region of the base of the vitreous. This vascularized mesoderm is definitely internal to the retina. It is from this tissue, which resembles angiomatous tissue, that the hemorrhages and subsequent organization and contraction stem.

The important embryologic feature is the adherence of this vitreous to the adjacent inner layer of the optic cup. Over this layer the secondary vitreous fails to form and, therefore, the retina is detached with further detachment from the subsequent hemorrhage and organization.

In 1946, Krause[10] reported 18 cases of what he termed encephalo-ophthalmic dysplasia. Sixteen globes were available for study. The retina was the tissue most commonly affected and was completely detached in 12. It then degenerated and atrophied. Posterior synechias formed. Proliferation of the glia, mesodermal connective tissue, and neuro-epithelium may lead to the formation of a transverse retrolental membrane and of iridic and postlental capillaries.

In 1949, Klien[29] reported the pathologic findings in three globes with retrolental fibroplasia, one patient having bilateral enucleation. She found complete axial detachment in all three eyes with marked subretinal hemorrhage in one. In all three eyes there was a fibrous membrane present in back of the lens. Between the face of the retina and the equator in each of the specimens was a structure which she thought represented remnants of the primary vitreous. In this area there were many newly formed blood vessels, which she believes were angiomatous in nature.

The pathologic lesions in both specimens (Cases 11 and 12) (fig. 6) were quite similar to the findings already reported. Both show marked axial detachment with presence

of true and pseudorosettes, and massive subretinal hemorrhage. In both there is a retrolental membrane present.

Similar to the reported cases, the most striking site of pathology is the area near the ora, immediately behind the equator of the lens. Here there is a mass of newly formed connective tissue and blood vessels which seem to involve not only the surface of the retina, but the retina itself. In fact, both cases show massive proliferation on the posterior retinal surface, in other words, under the retina. There are many newly formed vessels in this area and some old hemorrhage.

Another extremely interesting observation is the apparent proliferation and detachment of the epithelium of the ciliary body, which is thrown up into long fingerlike folds in this area of proliferation, seemingly detached by contraction of the membrane. Clinically, it has been stated that the ciliary processes can be seen through the ophthalmoscope when the mass is viewed in the extreme oblique position. These findings would suggest that possibly the pigmented areas are not truly the ciliary processes, but detached ciliary epithelium.

Terry[6] has listed the seven characteristic clinical findings: (1) Vascularized tissue behind the lens; (2) micro-ophthalmos; (3) shallow anterior chambers; (4) fetal blue irises; (5) ciliary process in the extreme periphery of the retrolental membrane; (6) searching nystagmus; and (7) an apparent photophobia. In addition, the disease is usually bilateral and essentially a disease of prematures.

On those grounds, our two cases are hardly typical clinically. Although both were definitely premature, one was unilateral, while the other had a localized macular lesion. However, both had typical anterior-segment lesions. In any case of retrolental fibroplasia with enucleation for suspected glioma, lack of the usual clinical findings would be expected. If the patient had the typical clinical picture, the eye probably would not have been enucleated as a pseudoglioma.

From the time of the first papers of Terry, in 1942, up to the present, the etiology and the pathogenesis of this condition have been the basis of much argument and controversy. The chief point at issue is whether this condition actually represents a congenital malformation or whether it is an acquired disease of the retina, probably metabolic.

In one of his last papers (1946) on the condition, Terry[7] still thought that the basis of the disease lay in the presence of a functioning hyaloid artery and a tunica vasculosa lentis in infants born prematurely.

In January, 1946, Reese and Payne[8] stated that they believed that Terry's retrolental fibroplasia and hyperplasia of the primary vitreous were one and the same thing. However, by 1949 Reese[9] had assumed the position that they are two very definite entities, the first one being similar to the old, well-accepted idea of persistent tunica vasculosa lentis (our TYPE I) and the second the condition which has now become known as retrolental fibroplasia. However, he states that both hyperplasia of the primary vitreous and retrolental fibroplasia have in common the persistence of the primary vitreous as the basic general lesion.

Klien[29] and Krause[10] also believe that this condition is essentially a congenital defect.

Recently Owens and Owens[11] have presented striking evidence that this is an acquired lesion and found that all of the premature infants they examined had apparently normal eyes at birth. On routine periodic examination of all prematures under three pounds they saw, 12.1 percent developed typical bilateral retrolental fibroplasia.

Earliest detectable abnormality was a slight dilatation of the retinal arteries and veins which was soon followed by one or more small, grayish elevations in the extreme periphery of the retina. Soon the margins of the disc became blurred and generalized retinal edema developed. The grayish, elevated masses in the periphery increased

in height, followed by the growth of a grayish membrane with numerous vessels from the periphery of the retrolental space, gradually forming a complete retrolental membrane.

The anterior chamber became shallow, the posterior synechia formed, and the iris developed atrophic appearance, and microphthalmos appeared.

These observations, of course, suggest that the lesion was acquired and possibly due to some environmental factor influencing the immature, metabolic, highly susceptible retina of the premature.

Recently in February, 1949, Kinsey and Zacharias[68] reported a large series of cases with retrolental fibroplasia to correlate this incidence with the many different environmental factors observed. They felt that there had been a very definite increase in the incidence of retrolental fibroplasia in recent years. They thought this increase correlates with the changes in the treatment of prematures, particularly the added vitamin-A supplement of the water-soluble type and of the increased use of iron.

In April, 1949, Owens and Owens,[12] in a very small series of cases discussed the trial of vitamin E in the prevention and in the treatment. They presented some clinical evidence that the use of high doses of vitamin E not only definitely decreased the incidence of retrolental fibroplasia, but in some early cases could cause a regression. They felt that iron and water-soluble vitamin A depressed the metabolism of vitamin E in a susceptible individual. Therefore, an excess of vitamin E was supplied with favorable results.

Recently Heath[69] attempted to correct some of this confusion by suggesting a classification based on gross pathologic grounds. He believed that a retrolental fibroplasia in its broadest meaning is a localized descriptive term for an ocular pathologic reaction of a nonspecific origin caused by disease in the patient. He has suggested a number of different groups, one of which is the retinal vascular group of prematurity.

Heath states that the fundamental pathologic picture of this type consists of edema, exudate of protein, hemorrhage, dilated capillary channels, and newly formed tissue. He thinks that there are three possible sources of this condition, the mother during gestation, susceptibility of the premature retina due to its structure and metabolism, and the postnatal infant care and environment before the eighth month.

He believes it is likely that the structural incompleteness and the unstable metabolism of prematurity are coupled with induced factors, such as deficiencies or excesses. The anterior loop of uncoapted retina lying over the ciliary body and sometimes over the ciliary processes is in the region of greatest growth activity. In this area there is both the unstable, fetal, anatomic matrix and the exaggerated metabolic needs of prematurity.

Although it is obvious that the concept of retrolental fibroplasia has been constantly changing since its inception and that much fundamental knowledge of the condition is lacking, it may be possible to reconcile some of the known facts.

The essential point at issue in the pathologic findings is whether the vessels and fibrous tissue in the area of the ora serrata represent angiomatous remnants of the persistent primary vitreous or whether the primary lesion is an intrinsic retinal reaction, possibly vascular, with secondary fibrosis and new-vessel formation.

Although it is obvious that no conclusions can be drawn from the examination of only two cases, it would appear that from these two patients retrolental fibroplasia is probably primarily a retinal lesion. Both of these cases are quite similar histologically in that in both there is retinal detachment with marked old subretinal hemorrhage. In both there is a proliferation of connective tissue and newly formed blood vessels in the area of retina near the ora. However, instead of

being in front of the retina, much of this proliferation is in the retina itself or even under the retina extending posteriorly along the inner surface of the detachment.

There is extremely little proliferation to form a retrolental membrane in either case. There is no evidence in either case of any angiomatous tissue in the area of the primary vitreous. This pathologic picture and the clinical observations of Owens and Owens[11] would seem to indicate that retrolental fibroplasia is primarily an intrinsic retinal disease, with the connective tissue and newly formed vessels a secondary reaction to the hemorrhage and inflammation.

From the standpoint of the pathogenesis, three factors seem applicable—prematurity, congenital defect, and environment. The exact site and type of the lesion in the retina might be determined by the concept of Heath; in the region of the loop of uncoapted retina in the ciliary body and ora serrata region, there is an unstable fetal anatomic matrix with the exaggerated metabolic needs of prematurity.

Falciform retinal folds seem to be a part of the retrolental fibroplasia syndrome as they are often seen in prematures with some of the typical anterior segment changes. These could represent a partial or segmental condition in which the lesion is present in only one area of the ora serrata region. Only in this area does the organization of fibrous tissue occur leading to a localized sector detachment.

This concept suggests a local defect might predetermine the area in which the condition could develop. This local developmental defect, possibly a vascular anomaly, could be due to a congenital lesion causing individual susceptibility. The actual exciting factor could be a metabolic trigger, such as suppression of vitamin E by iron and vitamin A, as suggested by Owens and Owens.[12] Thus, it is not only logical but possible that the background is a congenital local defect, that the site and the nature are due to prematurity, and that the active exciting cause is environmental.

Comment

Although it can be emphasized that pseudoglioma is a clinical concept made up of a number of fundamentally different pathologic conditions, it is interesting to note that there are some very definite interrelationships between the various groups.

As would be expected, all of these ocular conditions are definitely related to the immature age of the patient, as these lesions are extremely rare in adults. Only one type, the persistence of the posterior tunica vasculosa lentis, is a true developmental defect. However, the remaining types are characteristically found only in very young children.

Both retrolental fibroplasia and exudative retinitis are probably related to the metabolic peculiarities of the immature retina.

Chorioretinitis represented in this series by two cases of possible toxoplasma infection, probably an intrauterine infection, and vitreous abscess, probably secondary to meningitis, are infections typically found in the eyes of infants.

Massive retinal fibrosis has been described as occurring only in young children.

All of these lesions seem to be just as characteristic of infants and young children as is retinoblastoma.

The only clearly developmental lesion is persistence of the posterior tunica vasculosa lentis which is obviously due to an aberration in the usual regression of the anterior hyaloid system. Comparing this relatively simple concept of this condition to the complicated lesion of retrolental fibroplasia, it is becoming more difficult with increasing knowledge to accept the view that these two conditions are any more than very remotely related.

As suggested by Heath,[69] it is possible that exudative retinitis of the serofibrinous type of Leber and retrolental fibroplasia are related. It has been suggested above that both

of these conditions are primarily retinal lesions, possibly on a vascular metabolic basis. Retrolental fibroplasia is conditioned on the metabolic peculiarities of the immature retina of prematurity, while exudative retinitis of the Leber type is seen only in young children. As exudative retinitis of this variety is essentially unknown in the mature retina of adults, possibly the two lesions represent related reactions, the exact process depending on the state of maturity of the retina.

In summarizing the pathology of pseudoglioma in general, three types—exudative retinitis, retrolental fibroplasia, and massive retinal fibrosis—are fundamentally retinal lesions, while in organization of vitreous mass and in chorioretinitis, the retina is so massively involved secondarily as to make this an essential part of the condition. Only in one type—persistence of the posterior tunica vasculosa lentis—is the retina uninvolved.

The intraocular mass observed clinically is represented by: (1) A retrolental membrane, as in the persistence of the posterior tunica vasculosa lentis, the organization of vitreous masses, or retrolental fibroplasia; (2) a solid opaque detachment of retina, as in exudative retinitis, or chorioretinitis with inflammatory detachment; or (3) by a localized retinal mass as seen in massive retinal fibrosis.

In all of the 15 cases except four, the retina is completely and massively detached causing complete blindness. In the early case of exudative retinitis (Case 5) the detachment is localized to the posterior pole, but from our knowledge of this condition, it is certain that this would progress to complete detachment. The local retinal lesion in two cases of massive retinal fibrosis (Cases 3 and 15) appears to be active, but even without possible progress the useful vision in each of the eyes has been destroyed. In the case of persistence of the posterior tunica vasculosa lentis (Case 6), there was severe glaucoma of the developmental type, so that there was no possibility of any future vision in this eye.

Therefore, from the pathologic involvement present in each of these eyes, it is apparent that they were hopelessly blind and in several instances painful. Despite the possibility of tumor in each case, it is obvious that enucleation was indicated in each case.

(To be concluded)

I wish to thank Mr. William A. Moor of the Department of Ophthalmology, Washington University School of Medicine, for his efforts in producing the illustrations.

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SYNDROME OF GLAUCOMATO-CYCLITIC CRISES

Report of a case

Edwin Billet, M.D.
New York

Any case in which glaucoma is associated with cyclitis presents special problems in regard to diagnosis and therapy. Because of the heterogeneous nature of the cases grouped together as secondary glaucoma, therapy is more or less empiric and will vary with the past experience of the clinician.

It is possible to separate from this general classification of glaucomas associated with uveitis a relatively small group of cases which are characterized by uniformity in their clinical behavior and response to therapy. This group was described as a clinical entity by Posner and Schlossman[1] who named the syndrome of glaucomato-cyclitic crises.

The features of this syndrome as described by Posner and Schlossman may be briefly listed as follows:

1. The disease is unilateral.
2. The affected eye is white or may show a few dilated conjunctival vessels. Ciliary injection is absent.
3. The presenting symptom usually is slight discomfort, colored halos, or blurring of vision.
4. There is usually not more than a trace of aqueous flare. Glaucoma may appear a day or so before or simultaneously with the flare. Keratic precipitates may be present at the onset or may appear a day or two later. Posterior synechias never form.
5. Individual attacks last a few hours to one month, rarely over two weeks.
6. Episodes occur with varying frequency, without apparent cause.
7. The field of vision remains normal.
8. Provocative tests during symptom-free intervals are negative.
9. Treatment does not seem to shorten the course of the attack.

Posner and Schlossman state that surgical intervention does not prevent recurrence of attacks. Their statement, however, is based not on their personal experience but they cite Kraupa[2] who observed two cases with a similar clinical picture in which surgical intervention had no effect on the subsequent course.

The case here presented corroborates the fact that surgical intervention in the syndrome of glaucomato-cyclitic crises is ineffective. It serves also to emphasize the difficulty of differentiating this clinical entity from primary glaucoma on the one hand and from the general category of secondary glaucomas on the other. Finally, the current case indicates the beneficial effect of cortisone therapy in this syndrome.

Case report

M. L., a 50-year-old white man, a dentist, came under my observation on July 10, 1950. Beginning in 1943 and up to this date, he had had at least 15 well-defined acute attacks of glaucoma of his right eye, confirmed by tonometric readings. The longest attack lasted three weeks and was associated with a sense of fullness in the eye, colored halos around lights, and blurring of vision.

After the first acute attack a trephination of the right eye was performed in 1943 by another ophthalmologist. In view of continued recurrence of acute episodes, an iridencleisis was performed by still another ophthalmologist on the same eye in 1947. In 1948, a thorough medical investigation failed to disclose a definite etiologic agent. The left eye had never been affected.

During the six-month period of my observation, the patient had three well-defined acute glaucomato-cyclitic crises of the right eye. The left eye was normal throughout this period, maintaining an uncorrected

visual acuity of 20/20 and a tension of about 15 mm. Hg (Schiøtz).

First episode

The first episode began July 10, 1950, when the patient complained of a sense of fullness of the right eye of one day's duration, and a slight redness of the eye associated with rainbow halos. The vision in the right eye was 20/20 uncorrected and the tension was 37 mm. Hg (Schiøtz). Pilocarpine (one percent) and furmethide (10 percent) were alternated every hour during the waking hours. On July 11th, the tension was 31 mm. Hg and fresh unpigmented keratic precipitates were noted on the lower half of the right cornea. On July 12th, the tension was 25 mm. Hg, the eye was less red, and the patient felt considerably better.

The patient was symptom-free on July 13th, 14th, 15th, and 16th, during which period he was using pilocarpine (one percent) every four hours. On July 17th, while still using pilocarpine, the patient noticed that his right eye was becoming red again and felt full, and he was again aware of rainbow halos. Vision in the right eye was 20/40 and tension was 29 mm. Hg.

An aqueous flare and many fresh unpigmented keratic precipitates were seen. There were dense posterior synechias in the area of the coloboma; these probably were the result of previous surgery.

Neosynephrine (10 percent) was instilled without influencing either the tension or the other symptoms or signs during the next two days. On July 20th, the patient advised by telephone that he was much better.

On July 24th, the patient noticed that his right eye was again congested and felt full. The vision was 20/20, the eye was slightly injected and the tension was 34 mm. Hg. Homatropine (two percent) was instilled without any appreciable change in symptoms or signs. An aqueous flare was present and many fresh large unpigmented keratic precipitates were noted, as well as several pigmented ones. On July 26th, the tension in the right eye was still 34 mm. Hg and homatropine (four percent) was instilled. On July 27th, the eye was paler and the tension was 23 mm. Hg. On August 3rd, the eye showed no further signs of acute inflammation.

The patient was not seen again until August 28th when his vision was 20/20 in each eye and the tension was 11 mm. Hg in the right eye. Examination with the tangent screen during this glaucoma-free interval showed no evidence of central-field defect. This sharp drop in tension to below that of the other eye (15 mm. Hg) may be regarded as presumptive evidence for the complete recovery from this attack. The two partial remissions during the course of this attack are a part of the clinical picture.

Second episode

The second acute crisis began October 12th, manifested by some blurring and redness of the right eye and fullness. No medication was used during this exacerbation so that the effect of therapy might be better evaluated.

On October 13th, the vision in the right eye was 20/20 and the tension was 40 mm. Hg. There was an aqueous flare but there were no fresh keratic precipitates. On October 14th, the vision in the right eye was 20/25, there was more aqueous flare, and there were fresh keratic precipitates in the lower one third of the cornea.

On October 15th, the tension was 37 mm. Hg and the vision continued to be 20/25. On October 17th, the vision was 20/20 minus, the tension was 29 mm. Hg, there was less aqueous flare, and there were many fresh keratic precipitates. At this time the patient was feeling better.

On October 19th, the right eye was white, the vision was 20/20, the tension was 25 mm. Hg, there was no flare but there were many keratic precipitates. The patient was entirely comfortable. On October 21st, the tension was 13 mm. Hg, the vision was 20/20, the eye was white, there was no flare

but there were many residual keratic precipitates.

On September 2nd, between the first and second acute crises the patient felt a sense of fullness in his right eye with slight blurring of vision, but these symptoms subsided by the following morning without treatment. The patient was not seen at this time, but the history is suggestive of a mild attack.

Third episode

The third acute crisis began January 15, 1951. On awakening in the early morning, the patient felt deep pain in his right eye along with a sense of fullness in the eye, headache over the eye, and blurring of vision in the eye.

At three p.m., the vision in the right eye was 20/25 and the tension was 40 mm. Hg. A moderate aqueous flare was noted and there were many fresh keratic precipitates studding the posterior cornea.

Two drops of cortisone acetate (0.6 percent) were instilled in the right eye, every hour, for five hours. At eight p.m. the tension was 32 mm. Hg. While the aqueous flare was less marked, the subjective symptoms were unchanged. Topical cortisone therapy was continued without any other medication.

The following morning there was both subjective and objective improvement; the halos had disappeared and the tension showed a further reduction to 29 mm. Hg. The next day the patient was entirely symptom-free, the tension was 21 mm. Hg, and there were many discrete slightly pigmented keratic precipitates. The improvement continued, with the tension dropping to 18 mm. Hg on January 18th and 14 mm. Hg on January 20th.

Conclusions

1. A case of recurrent glaucomato-cyclitic crises is described. It is characterized by recurrent unilateral exacerbations of glaucoma and cyclitis.

2. Two filtering glaucoma operations had been performed without affecting the course of the disease.

3. The field of vision did not become impaired despite the surgical procedures and despite the 15 or more acute attacks of ocular hypertension, which varied in duration from several days to several weeks.

4. The first glaucomato-cyclitic crisis under observation lasted three weeks despite miotic and later mydriatic therapy.

The second crisis, during which all therapy was withheld, lasted nine days.

The third crisis under topical cortisone therapy lasted five days.

5. It is concluded that, possibly apart from cortisone, the usual therapeutic agents have no effect either on amelioration of a single crisis or on prevention of recurrent crises in this disease.

2625 Grand Concourse (58).

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RETROPLACEMENT OF THE INFERIOR OBLIQUE AT ITS SCLERAL INSERTION*

CONRAD BERENS, M.D., HELEN GRADY COLE, M.D., SOUREN CHAMICHIAN, M.D., AND MARJORIE V. ENOS
New York

Several years have passed since White[1] published his original observations on surgery of the inferior oblique at or near the insertion. At that time one of us (C. B.), in discussing White's paper, outlined his experiences with this operative technique and stressed the comparative ease with which the operation could be performed without detaching the lateral rectus.

Since then, a greater number of cases have been operated upon and this report includes an analysis of the results obtained in 97 cases (37 clinic patients and 60 private patients) in which findings are complete enough for statistical consideration.†

The purpose of this report is to review the anatomy and physiology of the inferior oblique, to discuss the preferred technique for performing retroplacement of the inferior oblique, and to analyze our results.

SURGICAL ANATOMY OF THE INFERIOR OBLIQUE

The following description of the anatomy of the inferior oblique is based on our own observations and those of others, especially Motais,[2] Howe,[3] Krewson,[4] and Fink.[5]

The inferior oblique is composed of striated muscle fibers only, its tendon varying from 0.0 to 1.5 mm. It arises from a small fossa in the maxillary bone immediately posterior to the orbital rim in the lower nasal quadrant of the orbit, just below the fossa for the lacrimal sac. The inferior oblique is the shortest of all the oculorotary muscles, averaging 37 mm. in length.

From its origin it follows the floor of the orbit, running backward and temporally, as far as the inferior rectus muscle. After crossing beneath the inferior rectus, the inferior oblique curves upward around the globe (in contact with it) and ascends as far as the horizontal meridian of the globe, in relation to the inner surface of the lateral rectus, where it is inserted into the sclera in a curved line, convexity upward, averaging 10 mm. in length.

The insertion of the inferior oblique is quite variable in relation to the optic nerve and to the horizontal meridian of the globe. In one instance Fink,[5] who studied 100 eyes, observed that the nasal border of the insertion encroached upon the optic nerve and in another extreme it was inserted 10 mm. temporal to it. On the average, the posterior edge of the inferior oblique is 4.26 mm. (94 percent) from the optic nerve (fig. 1), thus being in close relation to the fovea (2.2

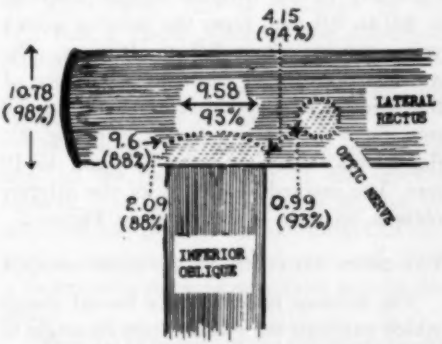

Fig. 1 (Berens, Cole, Chamichian, and Enos). Diagram illustrating the average measurements of the inferior oblique attachment from the optic nerve and the lateral rectus. (Courtesy of Dr. Walter H. Fink.)

* From the Department of Motor Anomalies, New York Eye and Ear Infirmary. Preparation of the manuscript was aided by grants from the John and Mary R. Markle Foundation and The Ophthalmological Foundation, Inc.

† Eighty-five retroplacement operations have been performed on 64 private patients and 90 retroplacement operations on 75 clinic patients to date.

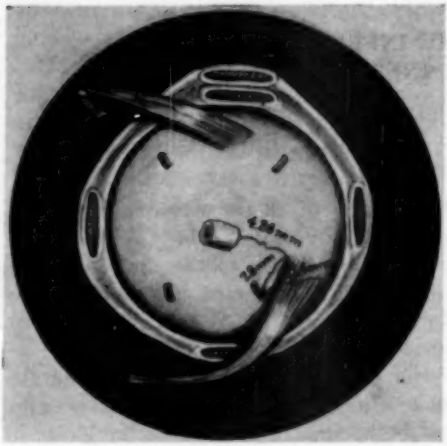

Fig. 2 (Berens, Cole, Chamichian, and Enos). Anatomy of the posterior half of the eyeball and average distance of the temporal edge of the inferior oblique insertion from the inferior temporal vortex vein based on seven adult eyes. (Cole.)

mm.). The curved insertion line usually encroaches upon the horizontal meridian but at times does not go as high as this.

The inferior temporal vortex vein is an important landmark during retroplacement operations. This vein emerges from the sclera at a level midway between the lateral and inferior meridians of the globe approximately 8.0 mm. behind the equator. The average distance of the temporal (anterior) extremity of the inferior oblique insertion is 8.0 to 9.0 mm. from the inferior vortex vein, varying from 8.0 to 16 mm. The average distance of the nasal extremity of the inferior oblique insertion averages 7.8 mm. from the inferior vortex vein (fig. 2), the distance varying from 4.0 mm. to 10 mm. The marked variability of the inferior oblique insertion is illustrated in Figure 3.

Fascia in relation to inferior oblique

The inferior oblique has a fascial sheath which envelops the muscle from its origin to its insertion. At the origin the sheath merges with the periosteum, and at the insertion with Tenon's capsule. At the inferior rectus crossing, the sheaths of the two muscles merge forming a common aponeurotic structure; however, there is more than a mere unification of the two sheaths at this junction. The sheaths assume a thicker, stronger, and more fibrotic character and numerous side bands re-enforce the union.

This strong and wide fascial expansion is called the ligament of Lockwood and supports the globe in a manner similar to a sling or a hammock. The ligament of Lockwood sends numerous fibrous expansions to the orbit from its external side and to the globe from its deeper side, constituting the check ligaments from the muscles concerned. It is also immediately connected with the sheaths of the other oculorotary muscles composing the intermuscular membrane.

Nerve supply of inferior oblique

The inferior oblique is supplied by the terminal fibers of the inferior branch of the third nerve. This nerve, after distributing branches to the medial and inferior recti, courses under the optic nerve and between the inferior and lateral recti to supply the motor branch to the ciliary ganglion. According to Fink,[5] the nerve then enters the ligament of Lockwood, at the point where the inferior oblique crosses the inferior rectus posteriorly, and seems to terminate 5.0 to 10 mm. beyond its entry into the body of the muscle. That there may be some degree of bilateral innervation from the third-nerve nuclei is suggested by Bing[6] and Whitnall.[7]

Fig. 3 (Berens, Cole, Chamichian, and Enos). Schematic representation of 15 variable positions of the inferior oblique attachment. Average distance from optic nerve (100 specimens) 4.26 mm. (94 percent). (Courtesy of Dr. Walter H. Fink.)

PHYSIOLOGY OF THE INFERIOR OBLIQUE

For the purpose of simplification, the globe will be considered to be capable of unlimited rotations around a practically fixed center of rotation. This center of rotation is the point at which the three major axes of the globe meet.

The movements of the eyeball are named in relation to the position of the center of the cornea as compared to its position in the primary position—that is, eyes fixing binocularly upon an object situated straight ahead at infinity.

Movements along the vertical axis of the globe move the center of the cornea outward (abduction) and inward (adduction). Movements along the horizontal axis elevate or depress the globe. Movements along the anteroposterior axis do not change the position of the center of the cornea but rotate a radius of the cornea clockwise or counterclockwise, this movement being called torsion.

The eyes are incapable of voluntary torsional movements and the only useful purpose in which torsion comes into play is in postural compensatory reflex movements. In normal binocular function the torsional effects resulting from oculorotary muscle action are so well counterbalanced by opposing muscles that torsional movements or tilted images are normally disregarded.

The primary action of the inferior oblique when contracted in the primary position is torsion. The line of action of the inferior oblique forms an angle of 51 degrees with the anteroposterior axis of the globe and 39 degrees with the horizontal axis. A muscle plane is that plane which includes the line of action of a muscle and the center of rotation of the globe. The greater the angle which a muscle plane makes with an axis, the more its action is distributed to movements along that axis.

The muscle plane of the inferior oblique makes a very small angle with the vertical axis of the globe, thus contributing very little to abduction or adduction which occurs around the vertical axis. The major portion is distributed to the other two axes, the larger share going to torsion, the ratio being approximately the same as 51 is to 39—that is, 56.6 percent affects extorsion and 43.4 percent affects elevation when the muscle is acting on the globe in the primary position.

As the globe is adducted (turned inward) the horizontal axis of the globe is turned more and more away from the inferior oblique muscle plane, thus the vertical component gaining in its share of the muscle action, when at an adduction of 51 degrees (which is not practical) the muscle becomes a pure elevator. When the globe is abducted from the primary position the contrary results and at an abduction of 39 degrees, the muscle becomes a pure extorter.

Theoretically, whenever the action of an oblique muscle is interfered with (as by a shortening or a lengthening operation) a large torsional variation is introduced. Practically, this is not apparent, and it has not interfered with the preoperative computations which are based only on vertical measurements since torsional measurements are not practical in many cases.

The important point is that a lesser vertical correction per millimeter of inferior oblique surgery is obtained than with surgery on a horizontal rotator per millimeter of intervention. This is explained by the fact that a major share of the difference introduced by the surgery affects the torsional movements as well, which are not compared pre- and postoperatively.

The computation and results are based only on the measurements of the vertical element. The normal relation of the line of action of the inferior oblique should be observed, preserving the 51-degree angle that it makes with the anteroposterior axis of the globe.

The nasal extremity of the insertion should be retroplaced less than the temporal extremity since the nasal edge is so close to the posterior pole of the globe. If the temporal edge is retroplaced 10 mm., the

nasal edge should be retroplaced approximately 8.0 mm. in order to keep the normal relations.

General considerations

White[1] has stated that "in all cases in which operation upon both inferior oblique muscles is indicated, recession of the muscles is carried out to equal or to unequal degrees. The amount of recession to be accomplished corresponds to experience in the recession of other muscles. The results were more accurately judged and more permanent when recession of the oblique muscles rather than tenotomy or myectomy was performed. When there is associated convergent or divergent strabismus, the external rectus may be prepared for resection or recession, but before the operation is completed the inferior oblique muscle can be easily engaged and recessed. This combined operation through the one conjunctival incision is proving increasingly satisfactory."

Dunnington[8] agrees that tenotomy and myectomy of the inferior oblique at its origin have produced inconsistent results, but suggests that the surgeon should be careful that the paralysis of the superior rectus is almost complete and that the upshoot is marked enough to warrant surgery of the inferior oblique.

The results of retroplacement have been more accurately judged and found to be more permanent than when a tenotomy or myectomy was performed. In the presence of the high lateral deviations (60^{Δ} or over), and when the vertical deviation is not excessive, it is advisable to correct the lateral deviation first so that the vertical element can be more accurately estimated to determine the amount of recession of the inferior oblique.

Indications for retroplacement of inferior oblique

This section deals with a most controversial aspect of muscle surgery.

Whenever an overaction of an inferior oblique seems to be a contributing factor in a case of binocular imbalance, every diagnostic method should be employed in order to determine the origin of the overaction, and every effort should be made to correct the defect especially when a functional result may be reasonably expected.

1. An overaction of an inferior oblique is present on the same side where the superior oblique of a nonfixing eye is paretic; that is, whenever the patient uses the sound eye for fixation, the unopposed inferior oblique tonus of the affected eye turns the globe upward. If the affected eye were to be used for fixation, the sound eye turns downward due to an overstimulated inferior rectus (yoke muscle of the superior oblique) on that side—following Hering's law, according to which a stimulation sent from the brain to one eye is equally distributed to both eyes.

2. When there is a paresis of a superior rectus and the affected eye is used for fixation, the inferior oblique of the other eye, the yoke muscle of the afflicted superior rectus, overacts and the sound eye shoots upward. In this instance the paretic superior rectus is receiving an overstimulation which is shared by the inferior oblique of the other side.

Although the overaction produced under the two headings just mentioned is only innervational in its origin, eventually spasm and contractures produce permanent organic changes in the constantly overacting muscle.

3. Anatomic anomalies, such as very loose check ligaments or abnormal insertions, may cause the muscle to overact. With the great variations possible in the insertion of the inferior oblique, it may be necessary to defer final diagnosis until the oblique muscle is exposed.

4. Normal overaction of the inferior oblique has been stressed by Duane[9] and more recently by Scobee[10] who attributes a normal overaction to the inferior oblique because of its mechanical advantages over its antagonist, the superior oblique.

These advantages, according to Scobee, are derived from the fact that (a) the effective portion of the inferior oblique is 37 mm. (the entire length of the muscle) as compared to the 20 mm. length of the superior oblique tendon; (b) the inferior oblique has a much larger arc of contact; and (c) makes a smaller angle with the anteroposterior axis of the globe.

Scobee adds "if both eyes are observed to upshoot in adduction, the condition is likely a normal overaction of the inferior obliques if the upshoot of each eye is approximately equal to that of the other. If, on the other hand, one eye upshoots more in adduction than does the other, then the condition is probably one of pathologic overaction of the inferior oblique and is due to an excessive innervation to a normal muscle or a normal innervation to a spastic muscle."

The importance of diagnosing a normal overacting inferior oblique lies in the fact that an inturning eye may show a hyper element but no vertical imbalance is present if the eyes are straight.

5. Primary spasm of the inferior oblique is a rare condition and difficult to diagnose as such. Duke-Elder[11] differentiates two types: (a) neurogenic, due to irritative conditions in the nervous system, and (b) myogenic, due to anatomic peculiarities of strength or insertion.

Contraindications

It is probably preferable to perform myotomy or myectomy of the inferior oblique if hyperphoria or hypertropia is greater than 10$^{\Delta}$ in the primary position, as measured with the cover test with prisms, unless, in addition, surgery of the superior oblique is to be performed.

Preoperative procedures

Examination

Before finally determining the desirability of operating on an inferior oblique, it is necessary to make as thorough an examination as possible. For example, the presence of binocularity in various fields of gaze should be determined and the possibility of fusion considered if it is absent. The major amblyoscope is invaluable for this purpose.

After the fixing eye has been determined, it should be noted whether the deviation is primary or secondary. The chart shown in Figure 4 has been designed to record the deviation in prism diopters in the main diagnostic positions of gaze.

The movements of the eyes are recorded from plus 4 to minus 4 to designate overaction or underaction with each eye fixing and the fixation used by the patient in the main diagnostic positions of gaze. The amount of correction necessary should be estimated for the deviation present with the normally fixing eye, irrespective of whether it is primary or secondary, that is, whether the sound or the paretic eye is fixing.

Several methods of examination and repeated tests are indicated in making a diagnosis, particularly when one is attempting to obtain a functional result.[12] Diplopia fields are often a great help in addition to version and duction studies, especially in phorias and cases of recent paresis.

Anesthesia

Local or general anesthesia may be administered.

Instruments

The necessary instruments for performing retroplacement of the inferior oblique are:

a. Eyelid speculum with solid blades
b. Bipronged scleral hook
c. Mousetooth forceps
d. Stevens scissors
e. Tenotomy hooks with long blades
f. Special muscle forceps*
g. Illuminated cupped spatula rectractor[13]
h. Measuring calipers
i. Needle holder

* Made by Storz Instrument Company, Saint Louis, Missouri.

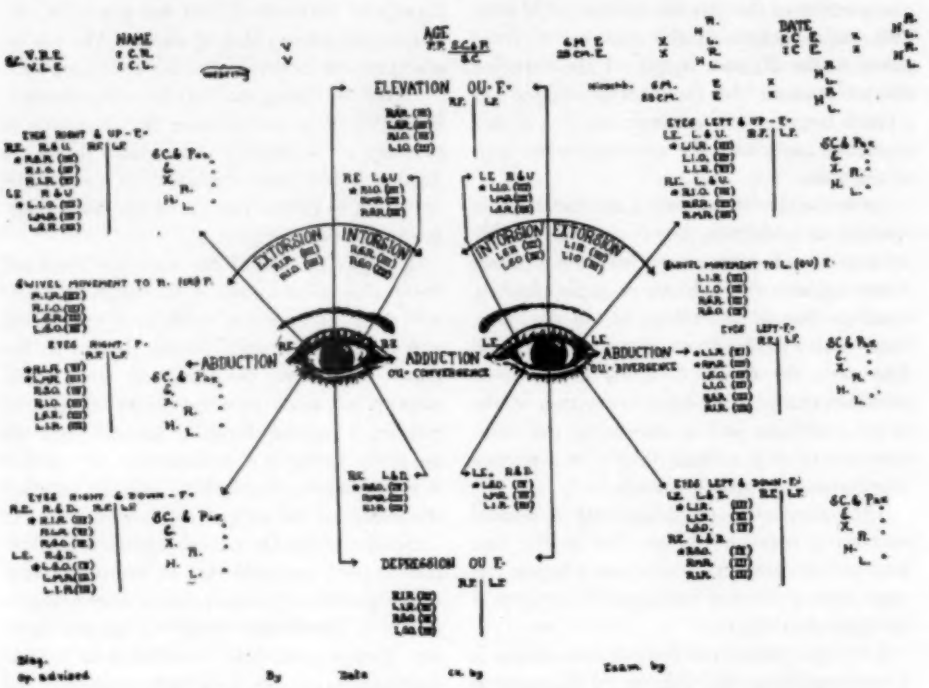

Fig. 4 (Berens, Cole, Chamichian, and Enos). Chart for recording fixing eye and relative motility of two eyes in the main diagnostic positions of gaze.

SUTURES:

a. Two double-armed blue nylon (5-0) sutures (D & G #P-443)

b. 5-0 plain catgut

TECHNIQUE WITH LATERAL RECTUS IN POSITION

First stage: Exposing the inferior oblique

1. The eyeball is rotated upward and nasally with a bipronged scleral hook or a black silk suture inserted in the episcleral tissue at the limbus.

2. An elliptical incision (convexity downward) is made in the conjunctiva, approximately 10 mm. from the limbus in the lower temporal quadrant of the eye.

3. A piece of Tenon's capsule is excised thus exposing the lateral and inferior recti muscles (fig. 5). By introducing a bipronged scleral hook and forcibly rotating the eyeball nasally and upward, while retracting the conjunctiva and Tenon's capsule with an illuminated plastic retractor, the insertion of the inferior oblique can usually be seen at the lower border of the lateral rectus.

4. The inferior oblique muscle and Tenon's capsule are grasped with fixation forceps at the insertion and carefully separated from the lateral rectus tissue. A strabismus hook may be passed beneath the muscle to draw it out of the wound, but, if this is done, care should be taken not to injure the inferior temporal vortex vein.

Second stage: Division of the scleral insertion of the muscle

1. The muscle is grasped with special muscle forceps passed over and under the muscle close to the scleral attachment. Blunt Stevens scissors are used to sever partially the tendon close to its insertion (fig. 6).

If traction is exerted on the muscle as the fibers are being severed and only small snips are made with the scissors, it is evident when the last fibers are cut and the danger of injuring the short posterior ciliary arteries and nerves is slight. However, Fink has an anatomic preparation in which the optic nerve is partially surrounded by the inferior oblique and this anomaly, although probably rare, should be kept in mind as a point in favor of incising the muscle close to but not at the insertion as first described by White.[1]

For the sake of uniformity, we have made it a practice to sever all capsular attachments of the inferior oblique to the lateral rectus. We have not severed the attachments to the inferior rectus and with small retroplacements (3.0 to 9.0 mm.), the procedure advocated by Guibor[14] would seem to be unnecessary and possibly undesirable. However, more information is needed on this point.

2. One or two (if the attachment was broad) double-armed white silk (4-0) sutures (D & G #P-521) were used in the early operations, just behind the muscle forceps through the muscle and capsule, and passed through the holes in the forceps.

a. After performing experimental operations on rabbits' muscles, cornea, and other tissues,[15] and finding that 5-0 blue nylon sutures* double-armed with atraumatic needles gave less reaction than any other suture we have used, we employed these nylon sutures in 134 operations for retroplacement of the inferior oblique. The two sutures are passed through the special muscle forceps which automatically produces exact spacing of the sutures after the muscle is freed from its scleral and lateral attachments (figs. 7 and 8).

b. At first when the lateral rectus was not reflected, it seemed advisable to mark the temporal extremity of the scleral insertion of the inferior oblique with a fine cautery, but

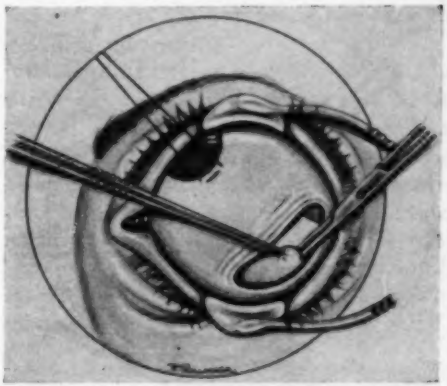

Fig. 5 (Berens, Cole, Chamichian, and Enos). Excising Tenon's capsule after making conjunctival incision.

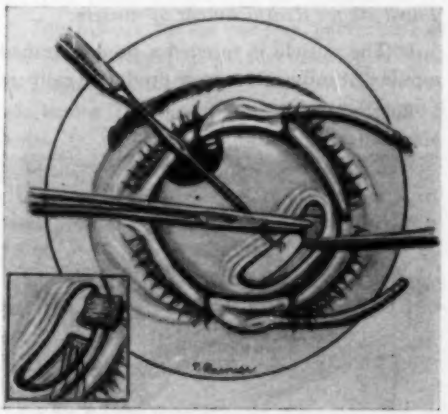

Fig. 6 (Berens, Cole, Chamichian, and Enos). Tendon of the inferior oblique is seized with forceps close to the anterior end of the insertion. The lateral rectus is in place.

INSERT: Retroplacement of the inferior oblique muscle after freeing the scleral attachment of the lateral rectus (later reattached by means of two double-armed (5-0) nylon sutures). This procedure simplifies the operation but is not necessary unless the lateral rectus is to be resected or receded.

* Produced by Davis & Geck for experimental purposes.

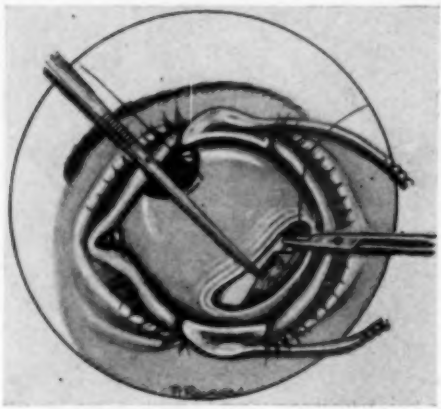

Fig. 7 (Berens, Cole, Chamichian, and Enos). Introduction of one needle of a (5-0) double-armed blue nylon suture into the freed end of the inferior oblique.

this procedure is no longer deemed necessary since the use of the bipronged hook and illuminated plastic retractor have made it possible to perform the operation with satisfactory visibility and comparative facility.

Third stage: Reattachment of muscle

1. The muscle is receded a predetermined number of millimeters measured with calipers (fig. 9—insert).

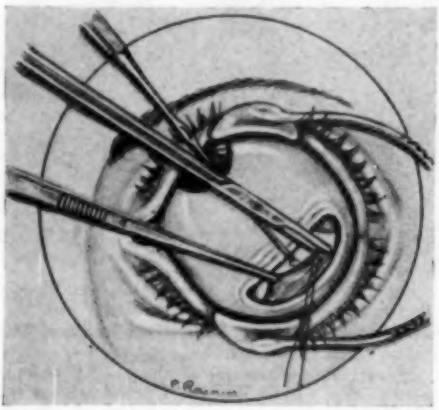

Fig. 8 (Berens, Cole, Chamichian, and Enos). Completing excision of the inferior oblique from scleral attachment.

a. In earlier operations when small retroplacements (under 7.0 mm.) were performed, it was found to be difficult to employ two sutures, and only one was used for a time. Since that time it became apparent to one of us (C. B.) that when two sutures were employed, the one placed near the optic nerve must be moved a shorter distance than the one placed at the temporal end of the

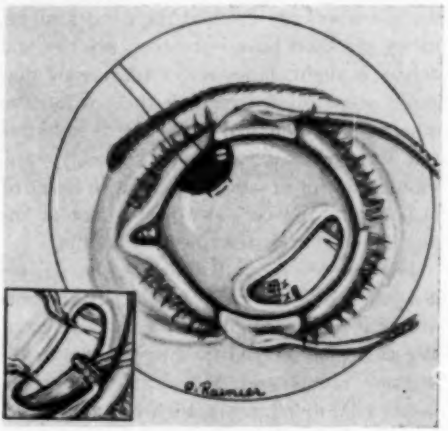

Fig. 9 (Berens, Cole, Chamichian, and Enos). Inferior oblique muscle sutured to sclera 10 mm. from insertion, usually one to two mm. nasal to inferior temporal vortex vein. Posterior suture should be placed nearer the original insertion to keep the new insertion parallel to it.

INSERT: Two double-armed sutures introduced showing forceps on muscle with two double-armed blue nylon sutures passed through it. Caliper marking point nasal to inferior temporal vortex vein for 10 mm. retroplacement of the inferior oblique. Temporal branch of caliper on nasal side of the insertion of the inferior oblique.

muscle if the proper anatomic relationships are to be preserved.

Furthermore, it was stated by one of us (C. B.) in discussing White's paper[1] that, when the needles were introduced 7.0 to 9.0 mm. from the insertion, one could easily injure the inferior temporal vortex vein, and the accuracy of this statement has been proved many times since then, both during operations and with the use of enucleated

eyes. Fink's anatomic findings also proved the correctness of this assertion.

2. The sutures are introduced into the episcleral tissues only, and tied with a surgeon's knot (fig. 9).

3. The conjunctival wound is closed with a running 5-0 plain catgut suture.

Postoperative treatment

Metaphen ointment (1:3,000) is applied to the lower cul-de-sac and eyelids. A light pressure dressing is applied and, if only one eye has been operated upon, a Ring's mask is used with a 15-mm. opening before the unaffected eye. The dressing is removed the following day and, if there is any appreciable reaction, cold saline compresses are applied. A perforated Ring's mask is continued as a splint.

Results of surgery of the inferior oblique

A compilation of the findings of a total of 97 patients (37 clinic patients and 60 private patients) with various amounts of retroplacement of the inferior oblique and the results obtained in the primary position at six meters, at 25 cm., and in the field of action of the inferior oblique, is shown in Table 1.

As it can be seen (table 1) from a comparison of the corrections obtained per millimeter of retroplacement, one cannot make an exact mathematical computation in order to correct the exact amount of an inferior oblique overaction. But this is also true concerning surgery on the horizontal ocular muscles. There are numerous factors which influence the amount of correction obtainable per millimeter of surgery and it is expected that the surgeon be cognizant of these factors.

One should observe especially, during the operation, the size, strength, and insertion of the muscle and at least gauge his correction to some extent upon these factors. Any profuse bleeding and traumatization will make the outcome much less predictable, due to resulting adhesions.

Because of the impossibility of precisely predicting results, the best we can do at present is to state that we attempt to correct one diopter of hypertropia in the primary position both distant and near by a retroplacement of one millimeter as a starting point and that approximately 2.0$^{\Delta}$ of correction is obtained in the field of action of the inferior oblique for 1.0 mm. of retroplacement.

Summary and conclusion

The surgical anatomy and physiology of the action of the inferior oblique muscle are described. There is great variability in the width, size, and shape of the insertion. The distance of the inferior temporal vortex vein from the temporal extremity of the inferior oblique insertion is variable—most frequently 9.0 mm. in 68 adult patients studied at operation. Tenon's capsule and the check ligament of the inferior oblique are described, as well as their nerve supply and physiology.

The history of the surgery performed at the scleral insertion of the inferior oblique is recounted and the technique of the retroplacement is defined, described, and discussed with its indications and contraindications. The postoperative treatment is given.

Our studies indicate that careful statistical data should be obtained concerning the effect of severing the attachment between the inferior rectus and inferior oblique. All the operations reported in this paper were performed without severing this attachment but all attachments to the lateral rectus were severed.

Presented is an analysis of the results of 97 retroplacement operations in which the preoperative and postoperative findings were sufficiently complete for tabulation. In 97 patients in whom an average retroplacement of the inferior oblique of 5.4 mm. was performed, an average correction of 5.0$^{\Delta}$ of hypertropia was accomplished in the primary

TABLE 1

AMOUNT OF CORRECTION OF DEVIATION IN PRISM DIOPTERS OBTAINED IN 97 PATIENTS BY CERTAIN NUMBER OF MILLIMETERS OF RETROPLACEMENT OF THE INFERIOR OBLIQUE

No. of Cases	Amount Retroplacement (in mm.)	Amount of Prism Diopters Corrected in: Primary at 6 Meters			Primary at 25 Cm.			Field of Action of Inferior Oblique			Fusion Status after Surgery: Grades							Length of Observation	Combined Surgery
		1*	2*	3*	1*	2*	3*	1*	2*	3*	1st	2nd	3rd	A.C.†	N	D.&N.§	?¶		
4	10	14Δ	18Δ	16Δ	14Δ	20Δ	18Δ	25Δ	43Δ	34Δ	0	0	1	0	0	0	3	1 to 2 yrs.	Resection L.S.R.—1 case
11	9	2Δ	20Δ	7.8Δ	2Δ	20Δ	9.2Δ	5Δ	32Δ	19.2Δ	1	1	4	0	1	2	2	7 mos. to 4 yrs	Resection R.L.R.—3 cases Retroplacement L.S.R. 3 cases—
14	8	1Δ	37Δ	7.8Δ	2Δ	43Δ	8.1Δ	8Δ	21Δ	14.3Δ	0	7	4	0	0	1	2	6 mos. to 3 yrs.	Retroplacement L.S.R. 1 case
13	7	1Δ	10Δ	4.2Δ	3Δ	12Δ	3.7Δ	6Δ	16Δ	10.9Δ	2	3	4	1	0	1	2	6 mos. to 4 yrs.	0
19	6	1Δ	18Δ	4.8Δ	2Δ	18Δ	5.2Δ	3Δ	18Δ	9.6Δ	3	3	9	2	0	0	2	6 mos. to 5 yrs.	Resection L.I.R.—1 case
12	5	1Δ	14Δ	4.6Δ	1Δ	12Δ	5Δ	2Δ	12Δ	6.7Δ	1	3	4	3	0	0	1	1 to 5 yrs.	Resection R.L.R.—1 case Resection L.I.R.—1 case
8	4	½Δ	4Δ	1.9Δ	½Δ	4Δ	1.3Δ	1Δ	10Δ	4.4Δ	1	1	4	1	0	0	1	9 mos. to 3 yrs.	Resection L.I.R.—1 case
2	3.5	2Δ	4Δ	3Δ	1Δ	3Δ	2Δ	8Δ	19Δ	12.5Δ	0	0	1	0	0	0	1	1 to 2 yrs.	0
9	3	1Δ	5Δ	2.2Δ	1Δ	3Δ	1.6Δ	4Δ	13Δ	8.1Δ	0	3	3	0	0	0	3	8 mos. to 3 yrs.	0
3	2.5	1½Δ	2Δ	2Δ	1Δ	1½Δ	0.5Δ	4Δ	9Δ	10Δ	0	0	3	0	0	0	0	4 mos. to 3 yrs.	0
2	2	?		1Δ	?	2Δ	1.7Δ	2Δ	9Δ	3Δ	0	0	1	0	0	0	0‖	9 mos. to 3 yrs.	0
Total 97	5.4			5Δ			5Δ			12.2Δ	8	21	38	7	1	4	17		
Ave.	1			0.9Δ			0.9Δ			2Δ									

1* Minimum amount of correction obtained.
2* Maximum amount of correction obtained.
3* Average amount of correction obtained.
† A.C. Anomalous Correspondence.
‡ N = Near.
§ D.&N. = Distance and Near.
¶ Fusion status not determined because of age or cooperation.
‖ One case had no fusion.

position for distance, 5.0$^{\Delta}$ for near, and 12.2$^{\Delta}$ in the field of action of the inferior oblique. One millimeter of recession obtained a correction of approximately 1.0$^{\Delta}$ of hypertropia in the primary position for distance and for near and 2.0$^{\Delta}$ in the field of action of the inferior oblique.

In the technique employed using two double-armed blue nylon sutures, the attachments to the lateral rectus were completely freed, but those to the inferior rectus were not severed. The posterior part of the muscle should be receded less than the anterior part.

Retroplacement operations which can be graduated are definitely preferred to myotomy and myectomy for lower degrees of hypertropia (under 10$^{\Delta}$ in the primary position). This assertion is made in spite of the fact that we have had excellent results with myectomy and myotomy near the orbital margin in patients who had only 3.0$^{\Delta}$ of hypertropia in the primary position. Certainly in the bilateral cases when the hypertropia is greater in one eye than in the other, retroplacement seems ideal.

708 Park Avenue (21).

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Ophthalmic Miniature

At supper the three Drs. of Physic again at my cabin; where I put Dr. Scarborough in mind of what I heard him say about the use of the eyes, which he owned, that children do, in every day's experience, look several ways with both their eyes, till custom teaches them otherwise. And that we do now see but with one eye, our eyes looking in parallel lines.

Pepys' Diary, May 24-25, 1660.

OBSERVATIONS ON VITAMIN DEFICIENCIES*

In an Eye, Ear, Nose, and Throat Clinic of a Japanese prison hospital

Max Edward Pohlman, M.D.
Los Angeles, California
AND
Edward Francis Ritter, Jr., M.D.†
Mattoon, Illinois

In view of the extensive reports on vitamin therapy in the literature, I thought it might be of interest to report observations on vitamin deficiencies seen in the eye, ear, nose, and throat clinic of a Japanese prison hospital, named Bilibid, for American prisoners of war.

Bilibid, located in the heart of Manila, was originally a Philippine prison and was converted by the Japanese after the fall of Corregidor into a prison hospital.

Bilibid prison consisted of a five-acre compound surrounded completely by a 20-foot stone wall, topped with high-tension wires, with sentry-manned observation posts approximately every 300 yards which made an escape virtually impossible. Inside the compound there were 15 80-foot long barracks with corrugated tin roofs and barred windows; these became the hospital wards.

The prison hospital was under the supervision of a captain in the Japanese medical corps who was later sentenced to 25 years' hard labor by the War Crimes Commission. From June, 1942, until October, 1944, Bilibid Prison was staffed by the United States Navy Medical Corps. From October, 1944, until February, 1945, when the hospital was liberated, the United States Army Medical Corps was in command.

Bilibid was the best equipped and supplied medical center in the Philippine Islands due to the fact that the Japanese permitted the Americans to use hospital and medical equipment that had been captured in Manila and on Corregidor. The hospital had other advantages in that there was electricity, an ample supply of potable water, and it was relatively rain proof.

Bilibid not only functioned as a hospital but also acted as a clearing house for all of the prison camps in the Philippines. Sick men from working parties would be sent to the hospital for treatment and be replaced by men from convalescent groups. The hospital census would vary from approximately 800 to 1,500 bed patients. The total census of the camp, because of incoming and outgoing drafts of men, would be at times over 3,000.

The eye, ear, nose, and throat clinic was remarkably well equipped in contrast to other prison hospitals. Daily sick call was held and usual treatments were carried out. More than 1,000 men were refracted in the clinic and fitted with lenses through coöperation of the Philippine Red Cross.

Xerophthalmia

The first serious eye complaint occurred in November, 1942, when 90 of the worst-looking human specimens imaginable were transferred from Cabanatuan (a prison camp in northern Luzon) to Bilibid for further treatment.

The men were all extremely emaciated, lousy, and suffering from deficiencies of all vitamins. Some of their pellagrous lesions resembled second-degree burns. However, they had been sent to Bilibid because of xerophthalmia with corneal ulceration. This was by no means the sum total of xerophthalmic cases at Cabanatuan, but merely the worst cases.

These men were placed on the highest

* Presented before the Kansas City Society of Ophthalmology, Otology, Rhinology, and Laryngology, March, 1951.

† Killed in action—China Sea—October, 1944.

caloric diet possible but symptomatic treatment proved to be ineffective as our supply of vitamins A and D was extremely low. At least 10 percent of the cases terminated with collapsed eyeballs from corneal rupture and others were developing dense leukomas as a result of the corneal ulceration. Fortunately, at this time we received our first Red Cross food and medical supplies and it was remarkable to watch the dramatic response of the eyes to proper food and vitamin therapy.

No new cases of xerophthalmia were seen after the arrival of the Red Cross medical supplies, as it was now possible to issue a multiple vitamin capsule daily to every man in the camp.

Optic neuritis

There were no further serious eye complaints until October, 1943, when we began to see our first cases of optic neuritis. By February, 1944, optic neuritis had reached epidemic proportions; approximately 90 percent of the camp inmates had some degree of optic neuritis.

The chief symptoms were photophobia, excessive lacrimation, severe retro-orbital pain and bitemporal headache, eye fatigue, and later loss of vision. At times the loss of vision was dramatic, I have seen cases go from 20/20 vision to counting of fingers in 10 days.

Physical examination showed no increase in intraocular pressure (approximately 40 cases were tested with the tonometer). The cornea was clear except for those who had previously had xerophthalmia. The media were clear. The retinal findings were so typical that we were able to classify them by grade.

Classification

Grade 1. There was a slight hyperemia of the retina but the disc margins were clear.

Grade 2. There was increased venous engorgement and hyperemia of the disc margins.

Grade 3. Typical choked disc, the entire fundus resembled a beefsteak.

Grade 4. There would be numerous flame-shaped hemorrhages and, occasionally, fluffy exudative reactions especially around the macular area. The more advanced and older cases would show a typical secondary optic atrophy with marked excavation of the disc.

Medical technicians were trained to take visual fields and, in no time, they became exceedingly proficient and proud of their work. I would estimate that at least 2,000 visual fields were taken by our men.

At first the visual field would show a constriction of the peripheral fields especially for the colors and an enlargement of the blindspot. Then small central and cecocentral scotomas would appear until eventually the blindspot and central scotoma would blend into one large scotoma.

Treatment

We felt that this eye compiaint was due to malnutrition and a vitamin deficiency so we named it, how accurately I do not know, beri-beri optic neuritis. Since there was no way to improve the diet, our main treatment consisted of massive dosage of vitamins. Our course consisted of:

First week. 50 mg. vitamin B_1, subcutaneously daily; 200 mg. vitamin C, subcutaneously daily; 100 mg. nicotinic acid by mouth; four vitamin-A and four vitamin-D capsules daily.

Second week. 20 mg. vitamin B_1 subcutaneously daily; 100 mg. vitamin C, subcutaneously daily; 200 mg. nicotinic acid by mouth; four vitamin-A and four vitamin-D capsules.

Third week. 10 mg. vitamin B_1, subcutaneously daily; 25 mg. vitamin C by mouth; 300 mg. nicotinic acid; four vitamin-A and four vitamin-D capsules.

Fourth to seventh week inclusive. 5.0 mg. vitamin B_1, daily orally; 25 mg. vitamin C orally; 200 mg. nicotinic acid; four vitamin-A and four vitamin-D capsules.

Following the intensive vitamin course, the visual fields would be retaken and the fundus reëxamined. If there were no im-

provement, or if the findings were worse, the vitamin course would be repeated.

In the acute cases, the patients would be atropinized for four weeks and would wear eyeshields made of cardboard with pinpoints for vision. Dilation of the pupils would usually relieve the complaint of severe bilateral temporal headache in the majority of the cases.

Efforts were made to eliminate tobacco as it was felt that it would further damage the inflamed optic nerve. However, despite our warnings of total blindness, we could not stop the men from smoking.

The intensive vitamin courses were not enough, however, the men also required a well-balanced diet that we were not able to furnish. I believe that vitamin therapy did prevent a great number of the men from developing optic atrophy, as some cases actually showed an improvement in the eyegrounds and visual fields after several courses of vitamins. The advanced cases of optic atrophy did not react at all.

Discussion

At the time we were seeing optic neuritis in epidemic proportions, it was interesting to me that very few patients complained of any ear symptoms such as tinnitus, hearing loss, or vertigo. Theoretically, the eighth nerve should have been just as sensitive to malnutrition and vitamin deficiencies as the optic nerve.

With this in mind, approximately 25 cases of advanced optic atrophy were studied from an ear standpoint. Hearing tests were made with tuning forks supplemented with the standard Navy whispered and spoken voice tests. Not one of these cases showed any appreciable hearing loss. Caloric tests with both hot and cold water on these individuals showed no remarkable change from the normal.

The same findings were obtained when 50 cases of acute, grade-4 optic neuritis were studied. Some of these individuals even suffered a dramatic loss in vision during the week they were being examined, yet showed no auditory changes.

Despite the extreme emotional and nervous strain of being imprisoned, with each day being a fight for survival, it is interesting that we did not see the acute labyrinthine reactions or Menièrelike syndromes that one would expect in such an environment. Possibly the low protein, low salt, rice diet may have been a factor. Other surveys in the camp revealed most of the men to have slow pulse rates with unusually low blood pressures.

A possible explanation for the greater resistance of the eighth nerve to malnutrition in contrast to that of the optic nerve was that noise at Bilibid was negligible. Maybe the number of nerve deafened would have equalled those with optic neuritis if the men had been imprisoned in a noisy place.

2200 West Third Street (5).

I would like to pay tribute to my colleague, Dr. Ritter, who was the ophthalmologist in our clinic and helped conduct this survey. Dr. Ritter lost his life when he and 1,800 other American prisoners of war were transported to Japan in an unmarked prison ship that was torpedoed by one of our submarines in the China Sea with five survivors.

AN AUTODEMONSTRATION OF THE "PHYSIOLOGIC NYSTAGMUS"*

GORDON L. WALLS, SC.D.
Berkeley, California

It has been known for decades that one's visual fixation is never as steady as one thinks it is. Fine fluctuations in the tonus-pattern of the extraocular muscles cause the visual axis to nutate about the point of regard, passing through that point only occasionally and accidentally.

The oculorotatory apparatus being a compound servomechanism, some sort of oscillation of the eyeball is actually inevitable; for, no automatic steering mechanism can hold a setting on "zero," or can even find zero, unless it is so damped as to be uselessly sluggish in its response to the need for a change of setting. In short, fixative reflexes could not be both prompt and precise—and it has been more necessary for them to be prompt than for them to be precise.

Even the innervations that produce the tiny oscillating movements of a fixing eye are "recorded" in the system, and are employed by the space-perceptual apparatus to compensate for the concurrent shiftings of the retinal image (Walls, 1951). The result is that the point of regard does not appear to oscillate. Subjective space stands as steady as if the physiologic nystagmus did not exist.

It may be said that the nystagmus was first postulated in the 19th century, for more than one investigator (Hoppe, von Fleischl) proposed such a thing in explanation of some minor visual phenomenon. Usually, only an oscillation in the horizontal meridian was conceived. Modern studies have shown that the horizontal movements are indeed the only important ones, for the suspected movements were at last recorded by Dodge in 1907, as an incident of his long program of study of eye-movements in general.

For a long time it was tacitly or overtly assumed that the physiologic nystagmus is undesirable—that is, that it must interfere with fine visual discriminations of various kinds.

An exactly opposite view was first taken by Andersen and Weymouth (1923). They were attempting to account for the fact that the thresholds found in measurements of "vernier" visual acuity are very much smaller than the resolution threshold, the reciprocal of which is the measure of ordinary visual acuity.

The vernier threshold, the binocular stereo threshold, and the angular threshold of visual movement are all truly spatial (whereas the process of resolution is generally regarded as a special case of intensity-discrimination [Walls, 1943]). The spatial thresholds are all of the same order of magnitude and, apart from this fact, it is quite easy to see that the three performances must have a single common physiologic basis.

In explaining the smallness of one kind of spatial threshold, Andersen and Weymouth were explaining all. An essential element of their theory was a fast, fine, scanning process in which the retinal image and the retina oscillated with respect to each other owing to the physiologic nystagmus. The effect of this was supposed to be an averaging of the directional signs of receptors receiving, at various moments, various parts of the diffraction image of any vertical line.

The subjective localization (directionalization) of the line was thereby made more precise than it could otherwise be. The visual field could be "chopped finer" than would be predicted from the center-to-center spacing of foveal cones conceived to be motionless.

The relative, oculocentric, directionalization of—say—the two lines of a vernier-acuity target, was liberated from this spacing, and the fact that the vernier threshold is only about one tenth of the diameter of a foveal cone was accounted for.

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* From the School of Optometry, University of California. Read before the annual spring meeting of the Western Section of the Association for Research in Ophthalmology, San Francisco, March, 1951.

More recently Marshall and Talbot (1942) have evolved a theory of the spatial acuities which is more complete (since more modern) than that of Weymouth and his students, but which similarly requires the physiologic nystagmus to exist and to have particular ranges of frequency and angular amplitude. Others (Adler and Fliegelman, 1934; Jones and Higgins, 1948; O'Brien and O'Brien, 1948; Blackwell, 1948; Horowitz, 1949) have discussed the pros and cons of whether the physiologic nystagmus should not also facilitate resolution performances.

In all of this time, however, there has been no direct experimental attack upon the question of whether any or all of the extensitive discriminations are actually helped or actually hindered by the unsteadiness of fixation. That is, no one has ever tried either to damp or prevent the nystagmus by mechanical immobilization of the eyeball, nor tried to exaggerate or alter the nystagmus by imposing a vibration upon the globe from an external source by way of (say) a contact lens, with measurements of acuities taken with and without these interferences.

Even the basic questions of the amplitudes and frequencies of the spontaneous movements remain unsettled. Dodge was the first to record a really rapid vibration of very small amplitude (50 to 63 per second; it showed plainly in his records but he did not quantify it; see Bartels, 1927); but it is rather the work of Adler and Fliegelman (1934) which has come to be considered "classic."

They found three rhythms of horizontal movement to be superimposed—that is, concurrent. The finest-fastest of these rhythms, and the one which was grist to the theoretic mill of Marshall and Talbot, had an amplitude averaging a little more than two minutes of arc and a frequency of 50 to 100 per second. Attempts, since, to find such fine and fast oscillations have been generally unsuccessful.

Gassowski and Nikolskaya (1941) found the fastest movements to be hardly oftener than two per second, with an amplitude averaging 10.5 minutes of arc.

Lord and Wright (1948; and see Lord, 1950) found little evidence for any such fast movements as were described by Adler and Fliegelman. In their two subjects there were movements at respectively about one-half second and two-thirds second intervals, through angles of three to 14 minutes; but these were not sinusoidal oscillations: each movement was a "flick" enduring only 0.02 to 0.03 seconds. Such movements are not the sort which would be "favorable" to spatial discrimination in terms of the Weymouth and Marshall-Talbot theories.

On the other hand, O'Brien and O'Brien (1948) deny that there are any movements with an amplitude as large as two minutes, and Hartridge and Thomson claim that the eyeball becomes motionless during periods of attentive fixation—this enabling Hartridge to make his well-known claim that different colored star-stimuli are fixated with different foveal cones.

Ratliff and Riggs (1950) have again found fine-fast horizontal movements, at a frequency of 30 to 70 per second and with a median amplitude of only 17.5 seconds of arc. The largest of these movements was not quite two minutes, and movements greater than one minute were "quite rare"; so, Ratliff and Riggs explain Lord and Wright's results as due to the low sensitivity (one minute) of their recording system. Moreover, they recalculated Adler and Fliegelman's data and found the amplitudes of their fine-fast movements to be only about one minute, not two or more.

Ratliff and Riggs also found horizontal movements of one to five minutes at two to five times per second, as well as slow "drifts" and rapid recovery-jerks. All of these grosser movements were ordinarily binocularly coordinated, the fine-fast ones never.* This

* Riggs and Ratliff: In press. The fine-fast movements were sometimes demonstrable in one eye when they were not in the other. Lord (1951) also finds the coarse "flicks" to be binocularly synchro-

suggests that the fine-fast movements are due to proprioceptive feedback from the muscles, going no higher than the cranial-nerve nuclei themselves.

In no one investigation to date have enough subjects been employed to reveal the extent to which individual variation may explain the differences in results. It, therefore, has to be said that we do not yet know how important the physiologic nystagmus may be, nor just what kind of importance it may have for or "against" visual-spatial discriminations.

Just for this reason, any new and simple means of studying the nystagmus automatically has some importance—while we wait for definitive investigations with elaborate equipment to disclose the true facts and resolve all the contradictions.

In the studies thus far published, intricate photographic apparatus has been involved, with the subject rendered most uncomfortable by the need for rigid fixation of the head and the provisions for reflecting an intense pencil of light-rays from a mirror or cup applied to the fibrous tunic (to furnish the necessary optical lever for magnifying the tiny eye-movements).

The phenomenon which has come to my notice is one which enables any individual to observe at least certain aspects of his own physiologic nystagmus continuously, to compare it under different conditions for the same eye, or to compare it in the two eyes successively or simultaneously. As a phenomenon, it is interesting enough in its own right to need no further excuse for a description of it; but, it is my hope that it may prove of value to the experimenters in this field, for screening purposes in the selection of suitable subjects, or for quickly obtaining large masses of crude-but-valuable data regarding binocular synchrony, relation to the laterality of ocular dominance, and the effects of convergence, of fatigue, and so forth.

To understand the manifestation I have found it is necessary to recall the effect known as the "Pulfrich phenomenon" and the physiologic basis generally assigned to it. If a pendulum or other reciprocating object, moving in a frontal plane, is viewed binocularly with a filter or other retinal-illumination-reducing device before one eye, the target is seen as moving in an orbit which is an ellipse with the minor diameter oriented "in depth."

The filter slows the transmission from retina to consciousness for the eye looking through it, so that at a given instant that eye perceives the target at a position behind the one (on the line of actual travel) in which it is seen by the other eye. The separate monocular directions of the target therefore intersect either behind or in front of the actual plane of travel, depending upon whether the target is moving rightward or leftward.

The basis of this, in the effect that a lowering of the intensity has upon the increase in visual response-time, can be elicited in monocular vision with apparatus no more elaborate than a cigarette.

Station yourself in a place so dim that you can barely see the white shaft of the cigarette when it is held upright at arm's length and shaken horizontally a couple of times per second through an amplitude of an inch or so. The glowing part will appear to be "loose," wobbling in jellylike fashion, upon the unburned portion of the cigarette. In any one movement, the bright coals can be seen to jump ahead, and lead in the movement, with the dim body of the cigarette catching up only when the hand comes to rest.*

nous. It should make a considerable difference, to the success of theories of the spatial acuities, whether the binocular movements are in phase, out of phase, or completely uncoördinated. The curious theory of Clark (1936)—a theory not of stereo acuity, but of stereoscopy itself—requires that the eyes oscillate in such a way that each is motionless when the other is moving.

* Just such a "slippage" of the bright parts of a shaken pattern, with respect to the dark parts, has been known for many years. Sanford (1903), for example, suggested the use of a grille of white paper strips on black velvet for seeing this slippage

A number of years ago I had several experiences in a theater which, before the performance, was illuminated only by elaborate filigreed luminaires attached to the side walls. Whenever I "steadily" fixated one of these, the whole constellation of bright apertures appeared to be detached from the dark body of the lamp, oscillating horizontally and continuously. Various ophthalmologists were sometimes with me, and also experienced the phenomenon; but none could account for it, nor did the explanation occur to me until I noticed the same phenomenon during a recent Christmas season:

My tree was decorated with "bubble lights" having bright, fluted basal globes. The numerous twigs of the tree formed an intricate pattern serving admirably as a framework in which to localize the lights. Moreover, because of the low reflectance of the foliage it was very dim as compared with the lights themselves.

When any one light was regarded, the whole group of lights oscillated horizontally as a unit, with respect to an apparently motionless tree. The average amplitude of the oscillation was quite easily estimated as four millimeters from a station four meters away. This angle (one mil) is much smaller than the amplitudes found by Adler and Fliegelman.† The estimated frequency agreed well with the findings of Lord and Wright.

To attribute the oscillation of the lights to a physiologic nystagmus of the eyeball may seem to contradict the earlier statement that the nystagmus does not cause apparent movements of objects. But here, the eye-movements are causing changes in the effective separation of the parts of each retinal image—the bright parts relative to the dim parts—owing to the effect of intensity-difference upon response time.

The alterations of relative oculocentric direction are therefore visible; but, all of the movement is attributed to one of the two sets of elements comprising the bright-and-dim pattern.

By closing the eyes alternately, it was easy to ascertain that the movements occurred similarly with each. With both eyes open, the amplitude seemed the same as with either eye alone. This was taken to mean that the two eyes were "in phase" for at least the greater part of the time. On the other hand, from an observation distance of only one meter the fluted portions of the lights appeared to be making tiny rotary oscillations. This would be expected if the movements of the two eyes were out of phase or independent, leading to depthy movements of the bright targets rather than to purely translatory movements in a frontal plane.

I leave it for others to explore this phenomenon and perhaps find serious applications for it. Its "subjectiveness" is its only fault, and its continuousness is its great virtue. The only approach to it that I know of is what has been called "Fox fixation": the development of the after-image of a bright ring, with which to surround a subsequent fixation-point so that any wobble of fixation will be evident to the observer himself. The after-image must of course be frequently renewed. The "Christmas-tree phenomenon" never runs down nor wears out!

which in his time was given very involved "psychologic" explanations.

† It needs pointing out that the whole of the true amplitude of the eye movement thus "seen" is certainly not seen: the amplitude observed will be a function of the intensity-ratio in the target configuration, and this ratio probably becomes optimal long before it becomes infinite.

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AN EVALUATION OF THE MASSACHUSETTS VISION TEST FOR VISUAL SCREENING OF SCHOOL CHILDREN

ELTON R. YASUNA, M.D., AND LEAH SANDERS GREEN, R.N.
Worcester, Massachusetts

Vision testing in schools has posed a problem of long standing. The National Association for the Prevention of Blindness stated recently in one of their publications,[1] "although comprehensive periodic eye examinations are the ideal, such a program is not feasible and periodic screening tests of vision are helpful in locating persons needing care. These, however, are gross tests serving only to indicate the probability, not the proof, of the need of eye care."

Until recently there has been almost exclusive use of the Snellen test which utilizes a chart with illiterate "E" symbols, numbers, or letters of the alphabet.

The Snellen test indicated those children displaying myopias, high refractive errors, and amblyopias due to various causes. It did not detect the hyperopic child, who might be having serious reading difficulties, or muscle defects such as tropias or high phorias.

Because of these serious limitations of the Snellen test in both scope and procedure, other tests were developed, among them the Massachusetts vision test. The test was first reported in a paper by Dr. Albert Sloane[2] of Boston.* This described a method of screening for hyperopia and muscle imbalance, in addition to distant vision. Dr.

*This paper was read before the Section of Ophthalmology at the 91st annual session of the American Medical Association in New York, June 13, 1940. It was based on the work done by a research group within the Division of Child Hygiene of the Massachusetts Department of Public Health. It was reprinted, with additions, from the Archives of Ophthalmology (**24**:924-939 (Nov.) 1940), copyright 1940, by the American Medical Association.

Sloane emphasized that his paper represented merely a preliminary report and was not to be considered as final.

Description of the test

The Massachusetts vision test consists of three parts:

The first is a measure of distant visual acuity, and a Snellen type illiterate "E" chart is utilized. The 20/20 line, consisting of six symbols, is used. Inability to read four of the six symbols correctly constitutes a failure.

The second portion of the Massachusetts vision test screens for hyperopia. Each eye is tested separately with a pair of spectacles, containing a plus 1.5D. sph. in each trial frame. Ability to read the 20/30 line is graded as partial failure and, if the 20/20 line is read, this is a complete failure. Two partial failures equal a total failure. If a child is able to read the 20/30 line with only one eye, thus scoring only one partial failure, then the test has not been failed.

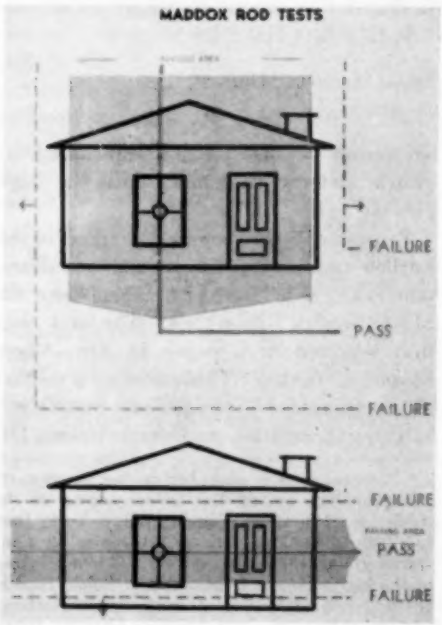

Fig. 1 (Yasuna and Green). The Maddox-rod test.

The last portion is for the detection of heterophoria. Vertical and horizontal phorias are examined by means of Maddox rods and a light at 20 feet. A picture of a window and a house is used, and so constructed that beyond the outer limits of house and windows is considered abnormal phorias.

The standards of failure are given as greater than 2^{Δ} of vertical imbalance, 6^{Δ} of esophoria, and 4^{Δ} of exophoria. Horizontal phorias are also tested at the near point, where the limits are set at 6^{Δ} of esophoria and 8^{Δ} of exophoria.

Whenever a child fails any portion of the examination, all further tests are omitted. As an example, failure to pass Part I (Snellen chart) would immediately stop further testing.

The illumination for the Massachusetts vision test consists of two rods of light for the illiterate "E" test, and a small bulb for the phoria test, both controlled by the examiner. The Council of Physical Medicine of the American Medical Association reported[2] that there were 11 foot-candles of illumination near the light source and six foot-candles of illumination at the center of the chart.

Since the original report[2] there has been no further published account of how the test has worked in practice. Much interest and discussion have been aroused, and many informal comments have been made to the effect that the test was "too complicated," "too strict," or "not strict enough." Therefore, it was felt that an analysis should be made of a carefully selected group in which all children were tested by one examiner and where close control of the group could be maintained.

The town of Shrewsbury, Massachusetts, was selected for study, and the following report deals with the findings and problems encountered in the use of the Massachusetts vision test in its schools during the school year 1949-1950.

Results

The entire school population of 1,575 students formed the experimental group (table 1). This included grades one through 12. Approximately 20 percent (307) failed and were referred for further ocular examination.

Of the 307 referred, 132 (43 percent) failed part-one (Snellen) of the test.

In the test for hyperopia (involving the wearing of plus spheres for Snellen Chart reading) 146 (48 percent) failed.

Twenty-nine pupils (nine percent) were referred because of failure to be within normal limits of phoria testing.

Notices were sent to the parents of each of the 307 students who failed the test. Enclosed in each notice was a form to be completed by the eye specialist of their choice. These findings were to be returned to the school. The notices did not furnish information as to which test was failed. This was done so that the eye specialists' findings would be the only criteria for treatment.

The form for the eye specialists requested a brief summary of significant findings, diagnosis, treatment, and suggestions, so that the school could better help the child. Reports were returned on 103 of the referred cases, or approximately one third.

It was possible to contact the parents of an additional 140 failures by telephone call or personal visit. Of this group so contacted, 58 had visited their eye specialists. Thus, of the original 307 referrals, answers were obtained in 243 cases (80 percent). Of this number, 161 are known to have sought the attention of the specialist, and their results can be evaluated.

TABLE 1

Results of Visual Screening at Shrewsbury

No. Tested	1,575	
Failures	307	
Part I (Snellen)	132	43%
Part II (plus spheres)	146	48%
Part III (phorias)	29	9%

Analysis of the Cases Followed

Results of Test I (Snellen)

Of the cases that could be contacted, 67 percent were seen by their eye specialist.

Seventy-five children were seen by their doctors because of failure in part one (Snellen) of the Massachusetts vision test. This group was subdivided into three categories by grades (table 2).

The first category included 13 children in grades 1, 2, and 3 (aged six through eight years). Ten received glasses and three were advised that no treatment was necessary. In most cases the correction was given for high degrees of hyperopic astigmatism. Of the three cases not receiving glasses, a pathologic condition which explained the amblyopia was noted. Correction was not considered to be of help in these instances.

The second group consisted of 26 children in grades 4, 5, and 6 (aged nine through 11 years), 18 of whom received glasses. The refractive error in all cases in which glasses were prescribed was myopia or myopic astig-

TABLE 2

Results in Test I (Snellen)

Age of Pupil (years)	No. Cases Studied	Glasses Prescribed		No Glasses Prescribed	
		No.	%	No.	%
6–8	13	10	77	3	13
9–11	26	18	69	8	31
12–18	36	33	92	3	8
Total	75	61	81	14	19

TABLE 3

RESULTS OF TEST II (PLUS SPHERES)

Age of Pupil (years)	No. Cases Studied	Glasses Prescribed		No Glasses Prescribed	
		No.	%	No.	%
6–8	28	9	32	19	68
9–11	16	8	50	8	50
12–18	28	19	68	9	32
Total	72	36	50	36	50

matism. No cases of hyperopic astigmatism were found. In the group of eight not receiving treatment, most had a pathologic condition which could not be corrected by lenses.

There were 36 children in the third group which represented grades 7 through 12 (aged 12 through 18 years). Thirty-three of the 36 received glasses and they were corrected in most instances for myopia or myopic astigmatism. Four were corrected for hyperopic astigmatism. Of the three cases not receiving treatment, each had an organic pathologic condition of the eye.

RESULTS OF TEST II (PLUS SPHERES)

The second group of failures included 72 cases. It was again considered best to subdivide the failures into three sections by grades (table 3).

Twenty-eight children in grades 1 through 3 (aged six to eight years) comprised the first group. Of this number only nine received glasses, leaving 19 cases in which no treatment was recommended by the specialist. All those receiving glasses were corrected for simple hyperopia.

TABLE 4

RESULTS OF TEST III (PHORIAS)

Age of Pupil	No. Cases Studied	Glasses Prescribed	No Glasses Prescribed
6–8	3	0	3
9–11	4	3	1
12–18	7	4	3
Total	14	7	7

In grades 4 through 6 (aged nine to 11 years) there were 16 cases. Eight of these pupils were given glasses, and the correction was about equally divided between simple hyperopia and hyperopic astigmatism.

The older age group (12 to 18 years) consisted of 28 children, and in 19 instances glasses were prescribed. Again, the correction was always for hyperopia and hyperopic astigmatism.

There were no comments by the eye specialists about the pathologic eye condition in those who were not given glasses.

RESULTS OF TEST III (PHORIAS)

There were only 14 cases in this section (table 4).

In the young age group (six to eight years) three cases were studied and none received glasses. One of these was reported as a high exophoria.

Three of the four failures in the middle age group (nine to 11 years) were given glasses after referral. Remarks of high exophoria or esophoria were included in these reports.

In the age group, 12 through 18 years, four of the seven cases were given a correction. Myopia was the causative factor in most instances, and one was reported to have had convergence insufficiency.

ANALYSIS OF RESULTS

Eighty-one percent of the failures in part one (Snellen) were given glasses. In all age groups the percentage was about the same (table 2).

Analysis of failures in part two (plus sphere) showed that exactly 50 percent of the children were given a correction. However, the glasses were given predominantly in the middle and older age groups. Only one third of the children in the lower age group (aged six through eight years) were advised to wear a correction (table 3).

There were only a small number of failures (14) referred and followed in part three (phorias). Although 50 percent of the children in this group were given glasses, there were none prescribed for any of the children in the youngest age group (aged six through eight years). All corrections were found in the middle and upper grades.

The prescriptions were always for refractive errors, and no remarks were found as to the use of prisms for muscle imbalance. Some of these children were advised to have orthoptic training.

Discussion and Recommendations

Part I (Snellen) of the Massachusetts vision test is valuable since 81 percent of the referrals were given a correction in the form of glasses. Although glasses in themselves are not the final criterion, they are probably the most practical gauge by which to judge whether a referral is worthwhile. In the remaining children for whom no glasses were prescribed, a good number showed a pathologic condition of the eyes, such as strabismus and optic atrophy.

In the testing of the young age group (six through eight years) it was found that many failed the 20/20 line but could read the 20/30 line. For this group the 20/30 line was used as the level of failure, although the directions for the test recommended the use of the 20/20 line. Otherwise the referral rate would have been much higher.

In the youngest pupils (grade one) both group and individual instruction on the illiterate "E" chart preceded the actual testing. This was found to be valuable and time-saving.

Although 50 percent of the failures in Part II (plus sphere) were given corrections, the results showed that most of the prescriptions were given to the older age group. For instance, in grades 7 through 12 about 70 percent were corrected, indicating a valuable test for mass examination.

In the nine-to-11-year ago group, a correction was given to 50 percent of the failures and, here too, it was felt that the rate was adequately high to warrant continuation of the test in its present form.

However, a different picture resulted in the analysis of the youngest age group (six to eight years). Here it was discovered that in 68 percent of the failures no correction or treatment was required at the time of examination. The specialists probably felt that students of this age could handle the degree of hyperopia found without the use of glasses.

Although the value of such a test was worthwhile in theory, nevertheless both the parents and the doctors were often left with the impression that the referrals were not required. Explanations about the need for the school testing were too frequently forgotten when the mother concluded that the visit and expense had been unnecessary.

In the directions for the plus-sphere test in the young, a +1.75D. sph. was recommended and used. A fewer number of referrals would result if a higher plus sphere was employed. A certain amount of hyperopia is present in children, and probably too many cases have been referred which might be considered near the normal for that age. Therefore, it is suggested that the strength of the plus sphere be increased for the younger children since a majority of them received no correction.

Sloane remarked in his preliminary report[2] that the strength of the plus sphere used was subject to controversy, and he stated, "in our hands it seems to be the 'happy medium.' It is likely that a stronger lens may be needed up to the age of 8 and 9 years."

Our results confirm this impression, and we would like to suggest the use of a

stronger plus sphere, such as a +2.25D. lens. Unfortunately, it cannot be assured that this new lens strength would be the ideal, and modifications may again have to be made if future tests show too high a rate of uncorrected referrals.

The failures resulting from the Maddox-rod testing for phorias were relatively few. Although it was difficult to come to definite conclusions on this small sample, certain impressions were received.

The test was easily understood by the older children. Sixty-three percent of the failing children, aged nine through 18 years, were given corrections. However, in the younger children, performing the test was both more difficult and time consuming, and the examiner was not always certain that the child had a good understanding of what was required.

Of the three children referred from the younger group, none was given correction, and once again the question of fruitless referral arose. It is suggested, therefore, that phoria testing with the Maddox rod be eliminated in grades 1 through 3, unless an individual problem should arise which would warrant this examination.

Wheeler, in discussing the initial presentation of the Massachusetts vision test,[2] emphasized the shortcomings of the Maddox rod in phoria testing and recommended that rapid screening be combined to increase the accuracy of the findings. He also objected to the suggested standards of phoria. Sloane felt that the use of an effective cover test, suggested by Wheeler, could not be administered properly by the average teacher.

All the failures found in the initial testing were reëxamined before referral notes were sent to the homes. The repeat examinations were performed by the school nurse, and this procedure is recommended.

Conclusions

The Massachusetts vision test has been found to be practical and valuable in mass screening for visual impairment in school children.

It is recommended that no testing should be done on the 20/20 line in Part I (Snellen) in the age group six to eight years, since too high a referral rate would result. Instead, the inability to read correctly four of the six symbols on the 20/30 line should be used as the basis of failure.

It is also recommended that the plus sphere employed in the age group, six to eight years, be increased from +1.75D. sph. to +2.25D. sph.

It is finally recommended that phoria testing with the Maddox rod be eliminated.

All failures should be rechecked by an independent examiner before referrals are made.

44 Pearl Street.

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MENIÈRE'S DISEASE ASSOCIATED WITH TRAUMATIC HYPHEMA AND GLAUCOMA

HARRY MCGRATH, M.D.
Bay Shore, New York

Menière's disease is a term which, according to Lindsay[1] is popularly applied to the recurring types of vertigo with fluctuating tinnitus and deafness. The confusion which until recent years was always associated with the interpretation of the so-called Menière's syndrome was due to the lack of evidence to establish the anatomicopathologic basis of the symptoms included in the syndrome.

In 1938, Hallpike and Cairns[2] found a hydrops of the labyrinth in two cases which showed the classical picture of vertigo, tinnitus, and deafness. Enough cases have now been studied pathologically to establish the fact that labyrinthine dropsy, a dilatation of the endolymphatic spaces of the inner ear, is the cause of Menière's disease.

Since the symptoms of this disease are of a recurrent nature and often paroxysmal in their onset, and since these symptoms are the result of hydrostatic changes in a cavity with a rigid wall, which is lined by a highly differentiated sensory epithelium with a rich vascular supply and filled with fluid, the analogy between it and glaucoma is obvious and striking. It is only natural that the term "glaucoma auriculare" has already been applied to this type of labyrinthine dropsy.

Although it is interesting to speculate that glaucoma and Menière's disease have many points in common in their symptoms, anatomy, and pathology, it is surprising that little attention has been given to the possibility that the two diseases might occur concurrently as the result of common etiologic factors.

There is no reference to any instance of glaucoma and Menière's disease occurring simultaneously in the same patient in the American literature or in any textbook. The only reference to the association of the two conditions in any language appeared in 1949 when Godtfredsen[3] of Copenhagen reported three cases of glaucoma with concurrent auditory-labyrinthine symptoms. In addition, he referred to three more instances which he had observed but not published.

Bearing in mind the observation that there are certain factors common to both Menière's syndrome and glaucoma, and that concurrence of the two diseases has been reported, the following case should be of interest.

CASE REPORT

J. S., a 52-year-old white man, was first seen on July 8, 1950. He stated that about an hour before examination, while he was working on a machine, a punch broke off and hit him over the right eye.

The injury was confined to the globe. There was no contusion of the bones of the orbit or skull. There was no loss of consciousness or any indication of any intracranial damage. The blow was not of a type which could conceivably have involved the inner ear directly. The skin of the lids was intact. There was deep bulbar congestion. The cornea was intact. The anterior chamber was filled to its horizontal diameter with blood. No fundus reflex was elicited.

Atropine was instilled and a pressure bandage applied. He complained of considerable pain in the eye, although the hyphema on the following day was only half the original height. The cornea was clear; the pupil was dilated following the use of atropine.

He did not return until July 12th, when he sought relief of intolerable pain, nausea, and vomiting. The bulbar conjunctiva was congested. The cornea showed no edema under slitlamp examination. The pupil was moderately dilated. The anterior chamber was not shallow and there was small hyphema below. Fundus details could not be seen clearly, although there was a faint fundus reflex. The intraocular pressure was 43 mm. Hg (Schiøtz) in the right eye, 20 mm. Hg in the left.

It was thought that the nausea and vomiting were natural concomitants of the eye injury and the increase in tension, but it was surprising to find these symptoms so alarming and prostrating that the patient had difficulty in maintaining his equilibrium. He was accordingly admitted to the hospital on the assumption that, under observation, repeated records of the intraocular pressure would show a rise more consistent with his symptoms.

The tension fell to 30 mm. Hg after the administration of eserine and pilocarpine and sedatives. Surprisingly, however, the nausea and vomiting became more severe, and it came to mind that they might have their origin in the ear rather than the eye.

Attention had been focused entirely on the eye, but questioning now showed that the patient had an annoying tinnitus in the left ear and to a less degree in the right, and that he had a sense of fullness in the left ear and could not hear as well as normally. When he sat up, there was a true vertigo, that is, a sense of rotation of surrounding objects.

He was confident that he had never had such attacks previously and the fact that he ascribed his complaint to his eye, and did not seem to recognize his symptoms as a disturbance of equilibrium, certainly pointed to lack of previous experience of vestibular involvement.

He stayed in the hospital for three days, during which time the eye became white and painless and the pressure returned to normal and was maintained at that level. The hyphema absorbed completely. The vertigo persisted with a little nausea but no further vomiting. The tinnitus was persistent and the fullness in the ears remained in spite of dehydration treatment, nicotinic acid, and dramamine. He was discharged from the hospital on July 16th. No drugs except those specifically mentioned were used.

On the following day a careful nose, ear, and throat examination showed that the ear drums were normal and the Eustachian tubes were patent. There was no evidence of any recent upper respiratory infection. Nasal ventilation was ample and the audiogram showed considerable loss in the high tones. Bone conduction was not increased and there was no lateralization to the Weber test. There was no nystagmus.

The visual acuity was now 20/30 in the injured eye, the tension was normal, and the fundus showed no abnormality. He was confident that there had been no previous attacks of vertigo and tinnitus, and his wife confirmed his statement. He was apparently well nourished and no signs of vitamin deficiency were disclosed. General physical examination showed essentially negative findings in the cardiovascular, alimentary, and central nervous systems.

For six months following the original study, there was no recurrence of the paroxysmal symptoms, but the tinnitus persisted. Repeated audiograms showed some fluctuation in the loss in the high tones. There was a constant sensation of unsteadiness but there was no vertigo except on sudden change of position. His work made it necessary for him to lie on his back beneath machinery. When he resumed the erect posture after lying flat, he always noted an alarming vertigo which was at times accompanied by vomiting.

He was last seen on December 7, 1950. The eye was quiet; vision 20/20; tension, 20 mm. Hg (Schiøtz). Peripheral and central fields were normal.

Comment

It is admitted that there is a distinct possibility of coincidence in the simultaneous development of glaucoma and hydrops of the labyrinth, or Menière's syndrome, in this single instance. Justification for reporting it lies in the fact that, for some unknown reason, simultaneous involvement of the eye and the ear in disease processes is far from rare.

Godtfredsen seems to have been the first to call attention to the frequency with which, in recent years, lesions affecting simultaneously the eye and internal ear have been observed. He notes that Granstrom and Nylen,[4] in 1938, published a report of Menière's syndrome associated with serous iritis which was at first hypertonic and later hypotonic. He then calls attention to various recently discovered syndromes, including congenital conditions due to infection in cases of toxoplasmosis and virus disease during pregnancy and the spino-ponto-cerebellar heredoataxias where labyrinthine deafness and retinal pigmentary dystrophy are important symptoms.

H. Sjøgren[5] is quoted as describing a new nosologic entity: reticular retinal dystrophy with concurrent defect of hearing. Godtfredsen does not mention the deafness associated with uveitis, vitiligo, poliosis, and alopecia in the syndrome known as Vogt-Koyanagi's disease, or the deafness which occurs with uveal infection in sympathetic ophthalmia and Harada's disease.

Carasquillo,[6] in an exhaustive survey of the Vogt-Koyanagi syndrome, notes that impairment of hearing is regarded as an integral part of the disease. He states that the concurrence of deafness with ocular disease was discussed by Peters[7] in 1912.

Snellen, in 1881, presented a case of sympathetic ophthalmia and deafness at the International Medical Congress in London. Peters cited six additional cases, including one of his own, in which such association had been established.

Mogan and Baumgartner,[8] in 1934, observed a case of recurrent interstitial keratitis of nonsyphilitic origin which was associated with labyrinthine symptoms. Cogan,[9] in 1945, reported four cases of a similar type of keratitis with vertigo and tinnitus and deafness and called attention to the frequency of its occurrence.

Godtfredsen seems to have been the first and only observer to have stressed the significance of the concurrence of glaucoma and labyrinthine disturbance and to have reported a series of cases to substantiate his theory that Menière's disease and glaucoma might have a common origin.

Godtfredsen's first case occurred in a man, aged 52 years. The eye condition is described as "glaucoma of the right eye" with "glaucomatous cupping, field defects and poor vision." One month following trephination there was a typical attack of Menière's disease with vertigo, vomiting, and nausea. The audiometer record showed high tone loss. The tension rose to 45 mm. Hg at the same time and subsided later.

In the second case there was "prodromal glaucoma" of the right eye for six years with normal fields and vision and no evidence of cupping of the disc. An acute Menière's attack occurred with a bilateral sudden rise of tension.

The third case was that of a woman, aged 66 years, of an "allergic, neurotic type." She had intermittent attacks of Menière's disease for 20 years. "Acute glaucoma" developed with vertigo and nystagmus. The eye was quiet for nine months following operation. The pressure rose later and the dizziness recurred.

Godtfredsen has also studied three other cases of glaucoma complicated by labyrinthine symptoms which are not detailed in his paper. He refers to all the cases he reports as "acute inflammatory glaucoma." In other words, the labyrinthine symptoms occurred in association with an acute congestive phase of the glaucoma.

In all three cases there were simultaneous rises in the intraocular pressure and attacks of vertigo. He notes that in two patients there were concurrent diseases—in one, ulcer of the stomach of a "neuro-vegetative origin"; while the other patient had "urticaria, vasomotor rhinitis, and was very nervous."

Menière's disease manifested itself six months and two years, respectively, after the glaucoma in two of his cases, and it occurred first in the third patient.

Godtfredsen feels that these neurovegetative lesions associated with the rise in intraocular pressure and hydrostatic changes in the labyrinth suggest a common vasomotor instability. He points out that Duke-Elder and Hess have recently stressed the angioneurotic element in primary glaucoma and that Mygind and Deterding[10] in Europe, and Atkinson in America, have postulated a similar origin of the labyrinthine disturbances in Menière's disease.

He thinks that secondary forms of acute glaucoma and Menière's disease may follow trauma and infections such as otitis media and iridocyclitis. The trauma or infection is said to liberate a histaminelike substance which is responsible both for the acute glaucoma and the labyrinthine hydrops.

In the case which I have presented, there is an obvious objection to the suggestion that the rise of pressure and the labyrinthine symptoms might have a common origin. There was extensive hyphema and, if this accounted for the rise of tension by mechanical blockage of the angle, there could by no stretch of the imagination be any connection between the glaucoma and any pathologic condition in the ear which gave rise to vertigo, tinnitus, and deafness. However, as Duke-Elder[11] points out, the etiology of traumatic glaucoma has aroused considerable speculation.

An intraocular hemorrhage may obstruct the filtration channels and accentuate rises of tension, but a traumatic glaucoma may develop in its absence and it is frequently associated with a normal or lower tension. The primary mechanism is probably, accord-

ing to Duke-Elder, an upset of the local nervous control of the circulation, any disturbance of which is generalized over the whole uveal tract by axon reflexes.

In my case, therefore, both the glaucoma and the Menière's syndrome could be explained as the result of liberation of H substance following trauma to the eye.

Little has been offered to explain the auditory symptoms associated with sympathetic ophthalmitis and the Vogt-Koyanagi syndrome. Calogero[12] suggested that the deafness occurring in sympathetic ophthalmia was due to irritation of the sympathetic nerves in the choroid. When these are stimulated the disturbance is transmitted to the labyrinth by way of the petrosal nerves.

On the other hand, Peters[7] explained it as the result of sensitization to uveal pigment. He was under the impression that the pigment in the basilar membrane was involved in an anaphylactic reaction. Koyanagi[13] and others doubted this theory for many reasons, particularly in view of the fact that there is normally no pigment in the basilar membrane.

Koyanagi thought that the deafness in the syndrome described by him was due to the action of a virus which also caused the uveitis. Hague[14] and others are convinced of the virus origin of the syndrome and therefore of the eighth-nerve involvement.

Cogan[9] does not suggest anything to account for the vestibulo-auditory symptoms which he described in association with keratitis. However, as cases of this syndrome have been reported in which section of the cervical sympathetic was practiced, there seems to be reason for considering a vasomotor disturbance as the cause of the labyrinthine symptoms.

If it is accepted that increased intraocular pressure and labyrinthine hydrops may result from common etiologic factors, this may be considered as evidence in favor of a neurovascular rather than a mechanical origin of glaucoma, particularly as the concurrence seems to be noted in acute narrow-angle types of glaucoma where the mechanical factor might seem to be beyond dispute.

Conclusion

The inferences to be drawn from the discussion of this single instance of glaucoma occurring concurrently with Menière's syndrome must naturally be in the main a paraphrase of those presented by Godtfredsen in his report of six cases of a similar syndrome.

"Menière's disease," as he remarks, "and acute inflammatory glaucoma are both comparatively rare diseases. Their association must therefore be considered a priori as an extremely rare phenomenon."

If further observations show that there is a definite syndrome of increased intraocular pressure, vertigo, tinnitus, and deafness, it should certainly substantiate the theory of a neurovascular rather than a mechanical origin of glaucoma.

In the case presented in this paper, it would provide evidence that, in traumatic glaucoma, neurovascular factors were responsible for the rise of pressure.

Nausea and vomiting are common symptoms of painful ocular conditions. In the case herein reported they had persisted for a considerable time before they were recognized as having their origin in the labyrinth.

If, behind the prostrating pain and nausea of acute glaucoma and other eye disease, the possibility of a labyrinthine disturbance is suspected, it seems likely that the concurrence of glaucoma and Menière's disease will be recognized as a syndrome and not a mere coincidence. Any instances of this association should be reported.

Summary

A case of glaucoma following blunt trauma with hyphema is presented. The nausea and vomiting which were considered to be due to the eye injury persisted when the eye became quiet. A typical Menière's syndrome—paroxysmal vertigo, deafness, and tinnitus—had developed.

There are many anatomic, physiologic, and

pathologic factors common to both glaucoma and Menière's disease, or hydrops of the labyrinth.

Six cases of glaucoma occurring concurrently with Menière's syndrome have been reported by one observer. It is possible that further observation will show that the two conditions have a common angioneurotic origin.

12 Shore Lane.

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LeCONTE'S BOOK ON "SIGHT"*

BEULAH CUSHMAN, M.D.

Chicago, Illinois

Dr. James W. White always referred to J. LeConte's book on "Sight" in his courses on the ocular muscles as the most satisfactory source for answers to the problems of monocular and binocular vision. It was written by a professor of geology at the University of California, and published by D. Appleton & Company, in 1881, as a part of a series called "International Scientific Series."

The author states in the preface that as a child he had been able to do many tricks with his eyes, and as he grew older he became interested in analyzing these actions according to existing theories of monocular and binocular vision.

Duane[1] in his article on "Binocular vision and projection," and Verhoeff[2,3] in "A theory of binocular perception," which appeared in 1902, and later in an article on "Effect on stereopsis produced by disparate retinal images of different luminosities," in 1933 and 1935, referred to LeConte's work and presented his own and similar ingenious experiments and explanations for the manifest squint patient.

LeConte, in this small book, gives many simple experiments for normal eyes. It is

*From the Department of Ophthalmology, Northwestern University Medical School.

easy and fascinating reading for the student of ocular movements. The book is so seldom referred to, and so few copies are available, that this rather extensive review was made for the students in the postgraduate class of the Department of Ophthalmology, Northwestern University Medical School. The students were also advised to read the book if they could obtain a copy. Its republication should be considered.

Review

LeConte described monocular as well as binocular vision, developing his ideas with many simple experiments. He states that a distinct image must be formed on the fovea by the optical instrument, including the lens apparatus, so arranged as to make the impression of light strong and definite by means of the image that falls on the nervous expansion of the retina which is specialized to respond to light vibrations.

Without images we would perceive light, but not objects, and the distinctness of objects is exactly proportional to the distinctness of retinal images.

Defects of eyes may be effects of misuse accumulated by inheritance for many generations. The range of the normal eye extends from infinity to five inches. Myopic eyes may range from a yard to four inches, from one foot to three inches, from six inches to two inches, or from three inches to one inch according to the degree of myopia.

Hyperopia is a structural defect, while presbyopia is a functional defect. The passive state for distance is normal but there is a loss of power to adjust for near objects.

At the macula lutea there is a finer organization than in any other part of the retina, and distinctness is the most perfect at this point.

To see a wide field, we unconsciously and rapidly sweep the line of sight over every part and then gather up the combined impression in the memory.

Distinctness of vision

Distinctness of vision is a product of the (1) optical apparatus and (2) retinal organization for distinct perception of the image thus formed.

Two forms of indistinctness are possible: (1) Those attributable to an imperfect retinal image, and (2) those attributable to imperfect retinal perception. The first is due to the optical instrument and the second to the organization of the sensitive plate of the retina.

Consensual adjustment of an eye is axial, with the focal adjustment for distinct vision dependent on an adjustment of the pupil.

Why do we not see the blindspot? The visual fields overlap and the area of distinct vision is very small. There is no bacillary layer in the area of the disc; therefore, there is no projection of blackness or darkness and thus an absence of visual acuity. The mind seems to extend the general ground color of the neighboring field of view over the blindspot. This is a psychologic rather than a visual phenomenon.

Images

An erect image is the result of the law of visible direction, the space in front of us is the outward projection of retinal states.

The internal retinal concave with all its states is projected outward and becomes the external spatial concave, and the two correspond point for point. Corresponding points, spatial and retinal, exchange with each other by impression and external projection. This gives the true position of all objects and radiants; therefore, it completely explains erect vision with inverted retinal images.

Binocular vision requires two eyes acting as one with visual fields and a field of vision, but we do see all objects double except under certain conditions.

Single vision is obtained when two images are projected outward to the same spot in space and, therefore, are superimposed and coincide.

Objects are seen as single when their retinal images fall on corresponding points of the retina. Each rod and cone in each retina has its correspondent in external space, and there they exchange with one another by impression and projection.

Objects beyond or nearer than the point of sight are always seen double, as their retinal images fall on noncorresponding points.

Double images formed by impressions on the two nasal halves of the retina are homonymous and those on the two temporal halves are heteronymous.

Horopter

Horopter is the name applied to the surface of single vision—that is, the surface passing through the point of sight, all objects lying in which are seen as single.

Instinct is an inherited experience. The property of corresponding points is partly inherited and partly acquired by individual experience.

The laws of projection give the true position of all points in space; this explains the apparent anomaly of erect images with inverted retinal images.

The law of corresponding points gives the coincidence of corresponding points in the two fields of view and, therefore, entirely explains the second anomaly of single vision with two retinal images. From the law of projection flow all the phenomena of monocular vision and, from the law of corresponding points, those of binocular vision.

The combination of similar images of two different objects may, therefore, produce the same visual effect as the combination of the two images of the same object. Single vision or ordinary perception of objects is by combination of two similar images, and it makes no difference whether the two images belong to the same object or two different but similar objects.

Perception of third dimension

The perception of the third dimension, depth or relative distance whether in a single object or in a scene, is the result of the successive combination of the different parts of the two dissimilar images of the object or the scene.

Stereoscopy, through the use of a stereoscope, facilitates the combination of two such pictures and the cutting off of the uncombined monocular images which tend to destroy the illusion.

Perspective

Three different forms of perspective are given: (1) Aerial—distant objects become dimmer and dimmer, bluer and bluer (helps in learning to judge distance); (2) mathematical perspective—objects become smaller and smaller and nearer and nearer together the farther away they are; (3) monocular or focal perspective—objects at the distance point of sight are distinct (the lens being adjusted for this distance), all objects beyond or within this distance are dim.

In binocular perspective, the eyes converge more strongly for near objects, less and less for more distant objects, with constant change back and forth by a kind of rapid almost unconscious triangulation; we, therefore, estimate the relative distance of objects in the field of view.

Binocular perspective operates perceptibly for great distances; mathematical and aerial perspective operate without limit.

The painter can imitate aerial perspective by diminishing brightness, dulling the sharpness of outlines, making blue the lines of all objects in proportion to their supposed distance. He can imitate mathematical perspective by diminishing the size of objects and the distance between them, but he cannot imitate focal perspective and still less binocular perspective. Therefore, the illusion is more complete and the perspective comes out more distinctly when one looks at a picture with only eye; whereas, in a natural scene, the perspective is far more perfect

with both eyes open because all forms of perspective coöperate.

To Wheatstone is due the credit of having discovered the fact that two slightly dissimilar pictures produce a visual effect similar to that produced by an actual solid object or an actual scene. He also invented the stereoscope to facilitate the combination of such pictures. But nothing can be more certain than that complete fusion never takes place.

Brücke's theory

Brücke's theory is that, in regarding a solid object or natural scene or two stereoscopic pictures in a stereoscope, the eyes are in incessant unconscious motion. The observer, by alternating greater and less convergence of the axis, combines successively the different parts of the two pictures as seen by the two eyes, and thus, by running the point of sight back and forth by trial, a distinct perception of perspective or binocular relief or depth of space between foreground and background is obtained.

Wheatstone repeats Dove's experiment to prove instantaneous perception of binocular relief, which Brücke's theory does not prove. Double images are necessary to binocular perspective.

LeConte's theory

LeConte's theory, hinted at by Helmholtz but not clearly demonstrated, is that "all objects or points of objects either beyond or nearer than the point of sight are doubled, the former homonymously and the latter heteronymously. The double images in the former case are united by less convergence, in the latter case by greater convergence of the optic axis.

"The eye (or the mind) instinctively distinguishes homonymous from heteronymous images referring the former to objects beyond, and the latter to objects this side of the point of sight. In the case of double images, each eye knows its own image although such knowledge does not emerge into distinct consciousness.

"Therefore, the mind perceives relief instantly and immediately for it does so by means of double images, and the relief is made clearer by a ranging of the point of sight back and forth, and a successive combination of the different parts of the object, scene, or picture, as maintained by Brücke."

Philosophy of vision

Images produced by a camera can be traced by mechanics, physics, and chemistry.

In the eye, the exquisite mechanism produces an image to a certain point beyond which we find something superphysical and chemical as at a certain point molecular and chemical change is replaced by sensation, perception, judgment, thought, and emotion. This part is incomprehensible.

Certain vibrations of the molecules of the brain, certain oxidations with the formation of carbonic acid, water, and urea here and there, appear on the other sensation, as consciousness, thoughts, desires, and volitions. They are as it were two sheets of blotting paper, pasted together; the one is the brain, the other the mind, as if certain ink scratches and ink blottings utterly meaningless on the one soak through and appear on the other as writing.

Outline of form is a combination of directions of component radiants.

Solid form is judged by binocular perspective and partly by shades of light.

Distance is judged by focal adjustment or monocular perspective; axial or binocular perspective by the amount of convergence necessary.

Judgment is regulated by intensity of light, color, and the direction of light.

Laws of parallel motion of the eyes are identical with Listing's law,[4] thus, "When the line of sight passes from the primary position to any other position, the angle of torsion of the eye in its second position is the same as if the eye had come to this second

position by turning about a fixed axis perpendicular both to the first and second position of the line of sight."

Laws of convergent motion

When the eyes move in the primary plane in the same direction (parallel motion) there is no torsion, but when they move in opposite directions as in convergence they rotate outward.

When the visual plane is elevated and the eyes move in the same direction by parallel motion, then lateral motion to the right produces torsion to the right, and to the left, torsion to the left; but when they move in opposite directions, as in convergence, then as the right eye moves left—that is, toward the nose—it rotates to the right, and as the left eye moves toward the nose—that is, to the right—it rotates to the left.

Therefore, when the eyes move in the same direction parallel to each other, as in ordinary vision of distant objects, then all their motions are governed by Listing's law; but when, to the contrary, they move in opposite directions, as in convergence, then the law of Listing is wholly abrogated, and another law reigns in its place.

The sum of all points which are seen as single while the point of sight remains unchanged is called the horopter. The horopter is the surface (if it be a surface) of single vision.

Thus, as in monocular vision, the more elaborate structure of the central spot restricts distinct vision to the visual line, and focal adjustment still further restricts it to a single point in the line, the point of sight; so also, in binocular vision, axial adjustment restricts single vision to the horopter, while rotation on the optic axis restricts the horopter to a single line.

In binocular vision with the optic axis parallel, as in looking at a distant object, the whole field of view and all objects in the field, including the visible parts of the face, are shifted by the right eye a half interocular space to the left, and by the left eye, the same distance to the right, without altering the relative position of parts, and the two eyes with their two visual lines seem to unite to form a single middle binocular eye and a single visual line along which the eye seems to look.

In convergence and divergence of the eyes, the two fields of view rotate in opposite directions, homonymously in the former case and heteronymously in the latter, about the optic center of the binocular eye, while the middle or binocular visual line maintains its position in the median line.

Verhoeff stated it well, when he wrote on "anomalous projection" and other visual phenomena, and in his article on "A theory of binocular perspective," that "two retinal images are not fused into a composite, but are replaced by a third identity, a compounded mean, and this function is a cerebral function."

25 East Washington Street (2).

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TRICHLORETHYLENE AS AN ANALGESIC IN OPHTHALMIC PRACTICE

BERTHA OFFENBACH,* M.D.
Boston, Massachusetts

This paper was inspired by the need for an analgesic suitable for use in the office examination of infants and young children who required fundus inspection and whose parents were unwilling or unable to have them hospitalized for general anesthesia. There was also need for a drug that would abolish pain, be easy to administer, be safe for the patient, and allow rapid recovery.

Trichlorethylene was found to be a most satisfactory agent for work with children, as well as in minor adult surgery in cases in which novocain and other analgesics were unsatisfactory. Trichlorethylene is reported here as a useful analgesic, *not* as a universal anesthetic agent. It is nonirritating and makes induction rapid and pleasant. It is less dangerous than chloroform and more potent as an analgesic.

"Trichlor" was brought to the attention of physicians in 1916 when four cases of bilateral paralysis of the trigeminal nerve were reported.[1] The patients were industrial workers exposed to trichlorethylene. The acute toxic symptoms of paralysis subsided in six weeks. Since the sensory division of the fifth nerve was involved, it was suggested that trichlorethylene might be used as a treatment for the pain of trigeminal neuralgia.[2] Herzberg[3] investigated the pharmacology of the drug and proved that it was nonirritating to the mucous membrane of the respiratory tract and caused no liver damage.

A high percentage of the patients with trigeminal neuralgia using trichlorethylene were relieved of their pain. In the beginning it was thought that the relief was a specific action of the drug on the trigeminal nerve. Later it was proved that the loss of pain was due to general analgesia; there is a relative degree of loss of sensation in various areas of the body and the trigeminal area responds early.[4] It is used today for tic douleuroux.

Trichlorethylene, a chlorinated hydrocarbon, is a colorless heavy liquid with the formula CCl_2:CHCl. It has an odor similar but distinguishable from chloroform. It is not inflammable nor will it explode when mixed with any proportion of air, oxygen, or nitrous oxide. Hence it is safe for use in electrocauterization. In strong sunlight it may decompose, so it is stored in a colored bottle. It is insoluble in water. Its action is thought to be by absorption into the blood stream, subsequently acting on the central nervous system. It is the least volatile of the inhalation analgesics.

In industry it is used in degreasing metals, in dry cleaning, and in the dewaxing of lubricating oils. Workmen have been seen leaning over the vats to inhale the vapors until they reach a mild stage of intoxication.[5] Fatal cases reported in industry from trichlorethylene have been due to the variety of impurities in the product.[6] There is a simple chemical test to show the presence of impurities.[7] The commercial product is called, "Triklone" or "Trethylene." The odor of purified trichlorethylene is different from that used commercially.

Histology of tissue taken from animals after prolonged administration of concentrated vapors of trichlorethylene showed no gross or microscopic changes in any tissue. Urine and sugar metabolism tests have shown no disturbances in patients using trichlorethylene as an analgesic. Tests show that it is less toxic than chloroform. British dentists prefer trichlorethylene to ether which has an irritating odor, and to chloroform which may cause liver damage.

I have used trichlorethylene in my office as an inhalant analgesic self-administered by

* Clinical assistant in ophthalmology, Massachusetts Eye and Ear Infirmary; ophthalmologist, New England Hospital.

the adult patient. Induction is rapid, usually in one-half to two minutes. A sponge is saturated with exactly one cc. of trichlorethylene and the patient instructed to hold it closely to his nose and inhale. After 20 to 30 seconds, he usually feels a sensation of warmth and tingling in the hands and face. Often there is a sense of euphoria. The patient is asked to report when he feels drowsy or dizzy.

At the point of drowsiness the operative procedure is begun and the analgesia continued. No indiscriminate dropping onto the sponge is done. The inhalant is exactly measured each time, being added to the sponge as needed in 0.5-cc. amounts. I have never used *more* than 5.0 cc. on one patient at one time. Recovery is almost instantaneous after the removal of the inhalant-soaked sponge. There are no after effects of nausea.

Fineberg[8] has noticed a perceptible lessening of surgical bleeding with trichlorethylene as compared to other analgesics.

The hesitation that many anesthetists have to using trichlorethylene would seem to be unjustified. The report of Waters[9] of 10 cases with cardiac complication after use of trichlorethylene included four cases in which electrocardiograms showed myocardiac damage before operation. Furthermore, trichlorethylene was used as a general, prolonged anesthetic in the second and third planes of anesthesia, beyond analgesia.

Case Reports

Trichlorethylene has been used as an analgesic in over 50 cases in adults and 25 in children when the ordinary drugs were contraindicated.

Mrs. A. D. was scheduled for removal of a chalazion. She admitted that she had reacted poorly to a previous injection of novocain given for dental work. Trichlorethylene was self-administered while the surgeon stood ready with instruments. One exactly measured cc. was dropped on the sponge and inhaled. When she affirmed that she felt tingling and drowsiness, the chalazion clamp was put in place and the incision made. An additional two cc. were dropped from a measure onto the sponge before the maneuvers were completed. The patient held the sponge herself. Recovery was rapid and left no unpleasant effects.

Trichlorethylene is useful as a diagnostic aid in headache problems. Functional headache can be diagnosed since it is eliminated when trichlorethylene is inhaled.

Mrs. E. G. complained of daily pain about the rim of the orbit, worse in the afternoon and evening. It often disturbed her sleep. A medical survey was negative. Examination of the area showed no tenderness, discoloration, or swelling. Intraocular examination was negative, as were field studies. The refraction findings were unchanged. Neurologic examination was negative.

Trichlorethylene was inhaled and, after 60 seconds, the headache had noticeably lessened. In three minutes she admitted her pain had left entirely. No headache was suffered for three days, at which time an additional application of trichlorethylene gave relief.

Trichlorethylene has been used by me in electrocautery procedures. There need be no fear of explosion in the presence of this drug. It is simple, safe, and effective during the removal of lid tumors and for Zeigler punctures in ectropion; to remove cataract sutures and for iodine scrubs in dendritic ulcers. It has been used in glaucoma patients who are habitual "squeezers." The relaxation is most helpful.

In young children, trichlorethylene induction is much the same as for adults except that the initial dose is 0.5 cc. This amount is held gently to the nose of the child. Many children enjoy the smell and willingly coöperate by taking deep breaths until drowsy. If there is resistance, the child is wrapped in a blanket. At the end of 30 seconds, the child becomes quiet and drowsy. Examination or manipulation is then done.

The dosage of trichlorethylene is increased by 0.5-cc. amounts as is required. Not more than a total of three cc. has been used for children under five years of age. No premedication has been found necessary. This analgesic is also used for children for fundus examinations, removal of a foreign body or chalazion, and for probing the nasolacrimal duct.

Baby G., aged four months, was brought to the

office showing a rhythmic head shaking, and nystagmus that had been present from birth. An attempt to examine the fundus was fruitless. Naturally, the child did not permit pressure on the globes to steady them, as one does with the adult.

The child sat upright in her mother's lap. A sponge, wet with 0.5 cc. of trichlorethylene, was held gently to her nose. She sniffed it, found it pleasant, and continued to inhale until the head-shaking slowed down at the end of one minute. In another 20 seconds the nystagmus had stopped so that a good view of the fundus was obtained. Two cc. of the drug were used, the child sitting up all the time.

Conclusion

Trichlorethylene is a recommended analgesic because it is bland and volatilizes poorly. It is safe for the noyice anesthetist. Patients recover instantly when the drug-saturated sponge is removed. They are able to walk home and have no unpleasant complication, such as nausea or vomiting. No patient has shown cardiac or other irregularities.

Since it ignites with difficulty and presents no explosion hazards, it is most useful when the electrocautery is used. It can be self-administered and assures a maximum of patient coöperation. There is a definite reduction in the amount of capillary oozing.

The inertia of trichlorethylene to combustion recommends its use wherever there is risk of fire, as in modern warfare where it might prove of particular value.

I wish to reaffirm that I am reporting the use of trichlorethylene as an *analgesic*; *not* as an *anesthetic*.

270 Commonwealth Avenue.

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Ophthalmic Miniature

The conjunctival irritations of the eyes, which are caused by smoke, over-heating, dust or similar injury, are easy to heal; the patient being advised first of all to avoid the irritating causes, as sun, smoke or the like; next, to bathe the eyes, in the beginning with lukewarm fresh water, later with cold, also to avoid bright light, and to keep the eyes closed. For the disease ceases without the use of any kind of medicine, if only a proper way of living be adopted.

Aëtius of Amida, *Tetrabiblon*, Chapter 3 (circa 535 A.D.),
From the translation by Hirschberg.

TRUE POLYCORIA*

NORMAN S. JAFFE, CAPT., (MC) U.S.A., AND
PAUL KNIE, 2ND LIEUT., (MSC) U.S.A.
Fort Campbell, Kentucky

The occurrence of more than one "real" pupil in an eye is so rare that its appearance merits considerable attention. The occurrence of accessory holes in the iris, on the other hand, is relatively frequent.

True polycoria may be said to exist when each pupil in an eye demonstrates a sphincter mechanism. The pupils must react to light and they must contract and dilate synchronously in response to drugs. Only four such cases have been previously reported. It is the purpose of this paper to add a fifth.

The term polycoria is carelessly used today to indicate all congenital holes in the iris. This includes iris dehiscences which are accessory slits in the iris not surrounded by sphincter muscle and iris diastases which are small holes at the root of the iris (congenital dialysis). These are best termed pseudopolycorias. In the latter there are often more than two holes. Von Hippel's[1] case had 16.

The mechanism of production of true polycoria is uncertain. However, several possibilities exist. The simplest mechanism is by the snaring off of a portion of the pupillary margin at a late stage, that is, after the sphincter muscle has formed. It is also possible for a coloboma of the iris to be separated from the true pupil by a bridge of tissue containing ectoderm (sphincter). However, the remainder of the coloboma usually has a pigment-layer border but no sphincter.

It may also be possible for any hole in the iris to obtain a sphincter mechanism if it develops about the time of differentiation of the sphincter from the ectoderm (65-mm. stage).

According to Coats,[2] another mechanism is possible. At first the cells of the surface of the gastrula embryonic stage are totipotential, that is, capable of differentiation into all the possible bodily tissues.

Later the cells become pluripotential, that is, capable of differentiation into all the tissues of a given organ only; not into all the possible tissues of the body. An abnormal differentiation of these pluripotential cells may occur anywhere so that it is possible to find smooth muscle on the posterior surface of the iris and other places. Usually, it is associated with pigment epithelium.

Pseudopolycoria, according to Mann,[3] may be associated with other defects in the same or other eye, such as small anterior synechias, typical coloboma iridis, corneal opacities, and capsular cataract in the region of the hole.

Dehiscence and diastasis may occur in the same eye. The cause of dehiscence is thought to be a mesodermal failure. In cases with a capsular cataract there was probably an adhesion of the mesodermal iris to the capsule of the lens, thus hampering the proper laying down of ectodermal iris. If the mesoderm separates later, it may be too late for the hole to close.

Also, if there is defective development of the mesodermal iris, or if there is atrophy of the latter, the ectoderm may split because of lack of support and this completes the hole. Mann[4] considers that fetal iritis is a doubtful cause of these adhesions and atrophies.

Probably the principal cause of diastasis or congenital dialysis is intra-uterine or early postpartum trauma.

CASE REPORT

A 21-year-old Negro soldier was seen in the eye clinic at the U. S. Army Hospital, Fort Campbell, Kentucky (fig. 1).

Vision in the right eye was correctible to 20/70 with a +2.75D. cyl. ax. 105°; in the left eye, correctible to 20/60 with a +2.0D. cyl. ax. 80°. In

*From Eye, Ear, Nose, and Throat Clinic, United States Army Hospital.

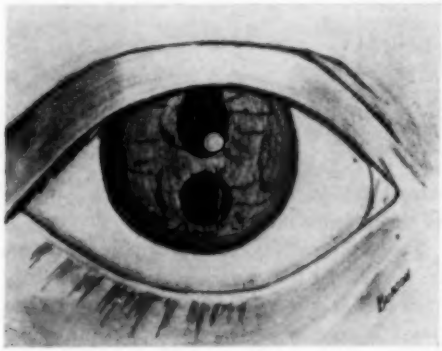

Fig. 1 (Jaffe and Knie). A case of true polycoria showing two pupils. Near the lower margin of the upper pupil an anterior polar cataract is visible. A strand of mesoderm runs vertically across the lower pupil. Because of the associated corneal opacities, photography was impossible.

spite of this defective vision, he was considered a good marksman.

Examination of the right eye revealed opacities of the cornea, most marked in the deepest layers and most dense in the central and lower central pupillary area. The opacities extended, however, in all directions. Adherent to the dense portion was a strand of mesodermal iris attached to the temporal portion of the iris at what would normally be the temporal midpoint level of the pupil.

Two pupils were present. One was directly above the other and each was shaped like a vertical ellipse. The upper one was slightly larger. It measured three mm. in height and two mm. in width. The lowest point was just below what would normally be the center of the pupil. The lower one measured 2.5 mm. in height and 2.5 mm. wide at its greatest width and was separated from the upper by a bridge of iris tissue about 1.5 mm. wide.

There were numerous, fine, persistent tags of pupillary membrane. One mm. above the lower margin of the upper pupil was an anterior polar reduplication cataract.

The left eye revealed a normal pupil, 4 mm. wide in a normally illuminated room. There were persistent tags of pupillary membrane, an anterior polar reduplication cataract in the center of the pupil, and corneal opacities similar to those found in the right eye.

Examination of the ocular muscles revealed the following: Esophoria for distance, 1Δ; esophoria for near, 6Δ.

Alternating hyperphoria: R.E., 4Δ; L.E., 8Δ.

Since it is impossible to demonstrate sphincter muscle histologically in the two pupils in the right eye, one must rely on reaction to various stimuli:

1. *Reaction to light.* Both pupils contracted briskly to light, both directly and consensually. A fine beam of light directed at the upper pupil caused contraction of both pupils as did a fine beam directed at the lower pupil. The contraction was as extensive and brisk as one would expect from a round, miotic pupil of about 2.5 mm. If one of these pupils did not contain a sphincter lining, contraction of the true pupil would tend to dilate the false pupil by iris traction. The left pupil reacted normally.

2. *Reaction to near.* The reaction was identical with the reaction to light.

3. *Reaction to 10-percent neosynephrine.* One drop instilled three times in the right eye at three-minute intervals caused a dilatation of the upper pupil to 7.0 mm. and the lower one to 5.5 mm.

The lower pupil was then seen to contain a vertical bridge of iris tissue running from the upper pupillary margin to the lower. This bridge actually divided the lower pupil into two pupils. The bridge was only a fraction of a millimeter thick. It was situated at the junction of the lateral two thirds and nasal one third of the lower pupil. If one of these pupils was a false one, dilatation of the true pupil would cause the false pupil to constrict passively.

4. *Reaction to pilocarpine.* One drop of one-percent pilocarpine was instilled three times into the right eye at three-minute intervals. Both pupils became very narrow with pinpoint dimensions. Again, contraction of a true pupil would cause a passive dilatation of a false pupil.

5. *Reaction to psychosensory stimulus.* Both pupils dilated briskly to a sudden clap of the hands. The corneal opacities made photographic material unsatisfactory.

Comment

The reactions of the two pupils in the right eye to light, drugs, and a psychosensory stimulus demonstrate conclusively that each is endowed with a sphincter mechanism.

The question of embryologic etiology excites considerable interest. Some insight into this problem is perhaps gained by an evaluation of the associated defects. Most important of these is the presence of anterior polar cataracts.

It is possible that a portion of the margin of the iris on both sides of the pupil became adherent to the anterior lens capsule. Subsequent separation of the mesoderm with union on the two sides would form the bridge between the two resultant pupils. If this occurs early enough, sphincter differentiation is possible in the bridge.

There is another possible explanation for the anterior polar cataract. The deep layers

of the cornea were opaque. Perhaps the commonest cause of developmental, noninflammatory corneal opacities is adherence between the posterior surface of the cornea and anterior lens capsule after separation of the lens vesicle from the surface ectoderm.

At this point the angle of anterior chamber has not yet differentiated and the chamber itself is practically nonexistent. This mechanism may well have been the operative one here since the left eye showed corneal opacities, anterior polar cataract, and no polycoria.

Ida Mann[3] states that associated ocular defects are relatively frequent with pseudopolycoria. However, in this case there are two pupils with excellent sphincter activity and associated defects.

The presence of a marked degree of alternating hyperphoria probably has no relationship to the polycoria. The patient never experienced diplopia probably because the lower pupil in the right eye was relatively occluded by the dense portion of the corneal opacities.

Summary

1. A case of true polycoria is presented.

2. There have only been four previous cases reported where each pupil had normal sphincter activity.

3. The case presented showed two pupils in the right eye, bilateral anterior polar cataracts, corneal opacities, and an alternating hyperphoria.

4. Reactions to light, drugs, and psychosensory stimuli demonstrated that each pupil of the right eye was associated with a sphincter muscle.

5. Differentiation from pseudopolycoria, as well as the possible mechanism operative in this case is likewise discussed.

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Ophthalmic Miniature

On examining the eyes I found both pupils contracted to little more than pin points, the right rather the smaller of the two. The irides were light-colored, and apparently healthy in structure. I could not observe any contraction of either pupil under the influence of light, but, on accommodating the eyes for a near object, both pupils contracted.

Douglas Argyll Robertson,
Edinburgh Medical Journal, **14**: pt. 2, 696, 1869.

NOTES, CASES, INSTRUMENTS

MOEBIUS'S SYNDROME

CONGENITAL OCULOFACIAL PARALYSIS: A CASE REPORT

J. R. STANSBURY, M.D.
Ventura, California

The occurrence of congenital paralysis of the horizontal rotator muscles of one or both eyes in conjunction with congenital paralysis of the facial muscles, unilateral or bilateral, seems to have been first described by Albrecht von Graefe in 1880.[1]

It was mentioned by Harlan in 1881 and Chisholm in 1882,[2] but Moebius in 1888, and again in 1892 when he reported 44 such cases,[3] described and classified the condition, and it is commonly referred to as Moebius's syndrome.

Moebius called the condition nuclear atrophy. Other and perhaps more descriptive names are: Congenital oculofacial paralysis or palsy (bilateral), congenital facial paralysis, and facial diplegia.

REVIEW OF LITERATURE

Features of the syndrome are not constant, but characteristically there is involvement of both external rectus muscles and the muscles of the face. In addition, in many cases, the adductor muscles are paralyzed, or at least paretic, resulting in little or no movement of the eyes in a horizontal plane, and the visual axes may or may not be parallel. The convergence reflex tends to be retained (although often diminished) even though voluntary adduction of either eye be impaired or absent.

Bonar and Owens[3] reported seven cases in which there was bilateral facial paralysis without involvement of ocular motion or of other cranial nerves, although in Moebius's series only those with unilateral facial involvement were free of ocular palsy.[3]

In addition to the facial muscles and the horizontal oculorotary muscles, there may be diminished or absent movement of the eyes in a vertical plane, as mentioned by Hicks,[4] and demonstrated in the case to be reported here.

Other and quite varied congenital deviations are frequently to be found in conjunction with the muscular palsies already mentioned. Henderson[5] reviewed 69 cases in 1939, all with seventh-nerve involvement and, of this group, 45 had an abducens palsy, which was bilateral in 43. Ophthalmoplegia externa was found in 15, lingual palsy in 18, ptosis in six, and trigeminal palsy in four. Club-foot was present in 19, branchial malformations in 13, there were pectoral muscle defects in eight, and six were mentally defective. Occasionally to be found is deafness, and the absence of vestibular responses is commonly present.[6] Deformity of the external ear,[2] webbed fingers or toes, supernumerary digits, defects of the neck and tongue muscles, and even absence of hands, fingers, feet, or toes have been reported.[6]

ETIOLOGY

From an etiologic standpoint, considerable uncertainty exists. Alcohol and syphilis play no part, and a history of antenatal disease or weakness is not common. A familial tendency has been noted on occasion, but the condition cannot be considered hereditary. Toxins, fetal inflammations, and encephalitis apparently are not involved and, although there are numerous associated malformations, nutritional deficiency is not a factor.[3] Males predominated in Henderson's[5] series but not significantly.

Kirby[7] gives two possible theoretical causes: (1) A primary aplasia of the muscle due to mesodermic defect, the embryonic foundation for the muscle not being laid down; (2) an ectodermic defect, the trouble lying in a failure of the normal connection between muscle and nerve, in improper development of the nerve, or in defective development of nerve nuclei.

In his case of abductor paralysis, esotropia

and right hypertropia, poor vestibular function, and diminished convergence, with paresis of the seventh and 12th nerves, Kirby was unable to rule out an encephalitis in infancy or a vascular injury to the corresponding areas of the brainstem, but felt that the peculiar distribution ruled against these possibilities, and that agenesis or abiotrophy of the sixth, seventh, and 12th nuclei was more likely.

Bonnevie[6] suggested that malformations of the cerebrospinal fluid system might be responsible for the development of the condition, and Murphy and German[1] reported a typical case in which encephalographic studies were thought to indicate hypoplasia of the metencephalon; there was also enlargement of the basal cisterns.

Pathology

Pathologically there is also some divergence of opinion. Early theories postulated aplasia of the brainstem in the region of the sixth and seventh nuclei and the vestibular pathway in the neighboring posterior longitudinal bundle (Kunn, 1895); a hypoplasia in this region (Oppenheim, 1895); and a localized fetal lesion (Moebius, 1895).[2]

Huebner (1900) sectioned the brainstem of a 19-month-old boy who had complete bilateral absence of abduction and paralysis of the facial musculature, and an atrophy of the left half of the tongue. He found marked defects in the motor ganglia and nerve fibers in the medulla and pons. There were practically no cells present in the nuclei of the facial, abducens, and left hypoglossal nerves. In the absence of signs of degeneration or inflammation, Huebner concluded that the defect was due to hypoplasia.[4] There was defective development in this case of the medial longitudinal fasciculus on each side, and the left olive and left pyramidal tract were poorly developed.[6]

Rainy and Fowler,[3] in 1903, showed atrophy of the nucleus and roots and trunk of the seventh nerve and concluded that, through lack of inherent vitality or defective nutrition, the cells perished, leaving evidence of their former existence in degenerated nerves.

In any case, pathologically there is agenesis of nuclei and nerve roots; cells of the nucleus are absent or diminished in number and abnormally small. Nerve roots are small or absent and, in some instances, the trunk is missing or of diminished size. The question of whether the primary process is one of "agenesis" or of "atrophy" seems a didactic one.

Clinical features

Clinical features of this syndrome tend to be quite variable, but the unchanging expression of facial nerve palsy, with open mouth and inability to close the eyes or whistle, are well known. The masklike facial expression may be aggravated by crying, and the position of the eyes depends on the extraocular muscles involved and the extent of their involvement—convergent strabismus may be present, but is not invariably found. The head turns on looking to the side, rather than the eyes, and excessive lacrimation occurs at times.

External ophthalmoplegia is rarely complete.[6] Visual acuity may be affected but usually is not. Conjunctivitis and corneal opacities may occur as a result of the inability to close the lids. Retention of the convergence reflex despite loss of voluntary adduction is typical, and attempted closure of the eyes results in Bell's phenomenon, with the corneas nearly disappearing beneath the upper lids as they move upward. Pupillary reactions tend to be normal.

A small, wrinkled, and atrophic tongue may be found,[6] and the mother may give a history of difficulty with nursing. The quality of speech tends to be altered but the mental capacity is more often normal than not. Club-foot is quite frequently present, and deficiencies of the chest musculature and even of the nipple[1] may occur. In the case cited by Murphy and German,[1] one-half of the face was rough and desquamating. Ab-

sence of extremities or digits, webbing, and supernumerary fingers or toes are occasionally found.

Vestibular testing by the caloric and turning methods may produce no nystagmus or other ocular movement. This finding indicates that blockage of vestibular impulses from involvement of the posterior longitudinal fasciculus prevents impulses from the vestibule from reaching the third nerve.

Hicks[4] observed that the occasional occurence of restricted vertical movement of the eyes must represent involvement of the motor nuclei far enough forward in the brainstem to affect at least portions of the third nerve. There must be only partial third nerve nuclear involvement, however, and fibers to the medial recti must be intact, otherwise the convergence reflex would be lost entirely.

When hearing is impaired, the process apparently extends to the eighth nerve also. Walsh[6] feels that the true Moebius syndrome is probably an intermediate condition between simple abduction paralysis and complete congenital external ophthalmoplegia.

Diagnosis

The differential diagnosis is relatively simple, being distinguished from progressive bulbar palsy by the fact that the latter is not present at birth and tends to be progressive in nature. Acquired facial paralysis, of the type due to obstetric trauma, is usually unilateral, unaccompanied by involvement of other cranial nerves, and transient.

The prognosis for life is good, but little improvement can be expected. Treatment, other than cosmetic, is of no avail.

Case report

History. E. M., a white boy, aged six years, was first seen in May, 1949. The mother had noted his waxy facial expression, convergent squint, and inability to close the eyes soon after birth. Birth of the patient, the third child, had been normal and spontaneous and followed an uncomplicated pregnancy and a five-hour labor at term. The two older children, a sister, aged 14 years, and a brother, aged 12 years, are apparently normal. There was no significant difficulty with nursing in infancy, but a club-foot on the right was noted early.

Past medical history revealed chickenpox at the age of three years, and measles and mumps at the age of five, but no serious illness or evidence of inherent constitutional weakness. A severe metatarsus varus developed on the right, and this was improved surgically to some extent in November, 1948, by plantar dissection and application of a long leg cast, to be followed later by bone surgery.

On examination, the boy was found to be mentally alert, coöperative, and physically quite sturdy and active. There was complete, bilateral, facial paralysis, except that the right corner of the mouth could be raised slightly with considerable effort, but he freely volunteered the information that he could not whistle, except through his teeth, and the forehead was typically smooth. Speech was rather thick and garbled, but otolaryngologic consultation revealed no defect of the palate, pharynx, or larynx, and the speech variation was felt possibly due to hypertrophic adenoids. Bell's phenomenon was present—the eyes rolled up and out on attempted (but unsuccessful) closure of the eyelids.

Ocular examination revealed normal pupillary reactions, and a mild squamous blepharitis. The uncorrected vision was: R.E., 20/40; L.E., 20/70; it was not appreciably improved with lenses, although cycloplegic (atropine) refraction revealed a considerable error of: R.E., +5.0D. sph.; L.E., +4.0 D. sph. ⊂ +1.0D. cyl. ax. 105°. (Vision is usually not affected in Moebius' syndrome.)

Slitlamp examination revealed parallel or concentric striations of the corneal epithelium of each eye which stained very superficially with one-percent aqueous mercurochrome or with fluorescin, and which were adequate to produce irregularities in the mires with the ophthalmometer. They produced no discomfort, however.

Peculiarly, the corneas seemed almost completely anesthetic, and there was partial insensibility of the bulbar conjunctivas. This would explain the absence of symptoms associated with deëpithelialization of the cornea due to corneal exposure in sleep, since the instillation of a bland oil at bedtime resulted in disappearance of the tendency to staining.

The remainder of the cornea, the anterior chamber, iris, and lens of each eye were normal microscopically. Ophthalmoscopic examination was entirely normal.

There was, in the primary position, a convergent strabismus of 15 to 20 degrees. He preferred to fix with the right eye (fig. 1). Horizontal motion was limited to not over 5 to 10 degrees with either eye. This was mostly confined to adduction, although with extreme effort a very slight degree of abduction was attainable with either eye. However, with the eyes maintaining essentially the same convergent relationship to each other, and being more or less fixed in the horizontal plane, the boy invariably turned his head in the indicated direction when instructed to look to right or left. Since he preferred fixation with the right eye, the head was

usually turned slightly to the right of the object viewed (fig. 1).

The convergence reflex was present but considerably impaired, the near point of convergence being at 12 to 15 cm. from the bridge of the nose, and the right eye tending to move up and in when the reflex was initiated. Considerably greater adduction of each eye was attainable by this means, however, than by voluntary conjugate lateral motion to right or left.

Vertical movement of each eye in a downward direction was normal, but upward motion was somewhat restricted, especially on the left. The nonfixing eye tended to move upward as the other eye took over fixation, as shown in Figure 1 (binocular hypertropia). Fixation with the left eye was slow, there was no fusion, and diplopia was not attainable.

Physical examination, except for hypertrophic tonsils and adenoids, dental caries, and the previously mentioned talipes equinovarus deformity of the right foot, was essentially normal. No paralysis or atrophy of the tongue or pectoral musculature was present (figs. 1 and 2).

Fig. 1 (Stansbury). Preoperative appearance with the right eye fixing. Note the smooth forehead, the normal tongue, left esotropia and hypertropia.

Fig. 2 (Stansbury). Appearance six weeks postoperatively. The corneal light reflexes are nearly centered, and the head is turned slightly to the right of fixation. Note normal pectoral musculature and varus deformity of the right foot.

Laboratory studies revealed nothing of significance, and the audiometric reading was normal, absolving the eighth nerve. However, no nystagmus or other ocular motion was elicited with caloric testing, indicating interruption of the vestibular reflex. Head-turning tests were not done.

Although it was felt that little could be accomplished from a functional standpoint, it was decided to try to improve the situation cosmetically, and the left eye was chosen as the initial site of attack, since it possessed the poorer vision.

On July 11, 1949, under general anesthesia, a somewhat modified Hummelsheim muscle transplantation was done on the left. The internal rectus muscle was first recessed 4.5 mm. behind its normal insertion. The external rectus was then exposed and cut one mm. from its insertion. The temporal halves of the superior and inferior recti were next

isolated and transplanted to the stump of the external rectus, which muscle was then resected for a distance of seven mm., and the cut end of its tendon reattached to its insertion without advancement.

In general, the four rectus muscles appeared to be of normal size, texture, and tone despite their improper motor innervation. Of interest was the finding of a two-mm. wide aberrant slip of tendon coming off the anterior surface of the superior oblique muscle to insert just behind and nasal to the insertion of the superior rectus muscle. This was not disturbed, and its significance, whether coincidental or in some way involved in the process, is not known.

From the time of the first dressing, the left eye has remained fixed in about five-degree abduction. No motion in the horizontal meridian was gained, and vertical motion was reduced by the surgical procedure to almost nil. However, the cosmetic appearance is quite satisfactory (fig. 2), with slight adduction on the right and slight abduction on the left resulting in what appears, on cursory inspection, to be parallelism of visual axes.

The head is held turned a bit to the right of the point of fixation. The convergence reflex was not affected. A small, transient, limbal corneal ulcer appeared a few days postoperatively, and produced no discomfort, again demonstrating the existence of an anesthetic cornea.

It is felt that nothing is to be gained by further surgery. At present the situation seems quite stable, with a satisfactory cosmetic result but without fusion or improved oculorotary function. The boy now seems to prefer to fix with the left eye instead of the right, which has some increased tendency to move up and in on attempted forced horizontal movement.

The corrected vision in the left eye is now 20/30, indicating the previous existence of amblyopia ex anopsia.

Tonsillectomy and adenoidectomy have been done (without much improvement in the quality of the voice), and further surgical repair is planned for the right foot. From the ocular standpoint, there seems nothing further to do except to protect the corneas from undue exposure. One wonders whether a simple recession-resection procedure, rather than the muscle transplantation, might not have achieved much the same cosmetic result, and preserved vertical motion.

Summary and conclusion

Congenital oculofacial palsy, known also as Moebius's syndrome, presents a wide latitude as regards location and degree of tissue involvement. The condition is a rare one, congenital, of uncertain etiology, but characterized pathologically by agenesis, aplasia, or atrophy of certain motor nuclei in the brainstem.

The nucleus of the seventh nerve is invariably involved; that of the sixth usually. In addition, there may be evidence of paralysis or paresis of muscles supplied by the third, fourth, fifth, eighth, 11th, and 12th cranial nerves.

Vestibular impulses may be interrupted through involvement of the posterior longitudinal fasciculus, but there is retention of the pupillary and convergence reactions. Other congenital stigmas, such as mental deficiency, club-foot, pectoral muscle defects, and deformities of the external ear and the extremities, may occur.

A case of Moebius's syndrome has been presented, demonstrating practically complete functional loss of the sixth and seventh cranial nerves with partial involvement of the third, probably the fourth, and unquestionably the fifth, as evidenced by corneal anesthesia. The sensory nerve loss is quite an unusual finding, appearing in only four of Henderson's[5] 69 cases. The case presented also shows interruption of vestibular responses and a unilateral club-foot. The eighth, 11th, and 12th cranial nerves appear to have escaped incorporation in the process.

Walsh's[6] conclusion that oculomotor involvement in this condition represents an intermediate step between simple abducens palsy (of the type due to a true developmental defect rather than to fibrosis of the lateral rectus muscle induced by trauma) and complete congenital external ophthalmoplegia seems inescapable. The underlying pathologic process remains, however, one whose cause and nature is unknown.

34 North Ash Street.

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PARADOXAL OCULAR MOTILITY AFTER CYCLOPLEGIA

JAMES MILES O'BRIEN, M.D.
Bridgeport, Connecticut

M. F., a four-year-old girl, was seen early in 1948, at which time a marked degree of concomitant strabismus was noted. The parents reported that the child's eyes had begun to turn in some two years previously. An ophthalmologist performed a refraction with drops, and the parents were advised at this time against any surgical procedure.

No history of squint was obtained from either parent, nor was it present in any of the other siblings. The obstetrical history was completely negative and the postpartum condition of the child was normal. No central nervous system involvement was manifest either during or after delivery.

When she was first examined, external examination, slitlamp examination, and examination of the fundi of both eyes revealed no pathologic changes. Vision was: O.D., with a +1.0D., 6/20; O.S., with a +1.5D., 6/20. The cover test showed a concomitant esotropia of 20 degrees; the synoptophore, 19 degrees to stop motion.

Prism and after-image tests revealed normal retinal correspondence. First degree fusion was obtained on the synoptophore at 18 degrees.

There was a slight upshoot of the left eye noted on cover of the right eye. However, it was not felt that a definite paresis was present but that the upshoot was a consequence of the extreme degree of inturning. The perimeter angle was found to be 20 degrees with the right eye fixing.

Measurements for near were taken on the perimeter arm at a distance of 33 cm. Measurements for distance were taken on the synoptophore. Fusional amplitudes were not measured due to the age and poor coöperation of the patient. All measurements herein recorded refer to esotropia.

An atropine refraction, performed following the use of atropine (one percent) for four days, revealed:

O.D., +3.75D. sph. ⊃ −1.0D. cyl. ax. 165° = 6/9; O.S., +4.0D. sph. ⊃ −1.25D. cyl. ax. 165° = 6/10.

The squint now measured:

WITHOUT GLASSES

Perimeter	27°
Cover	25°
Synoptophore	26°

WITH GLASSES

Perimeter	26°
Cover	25°
Synoptophore	23°

The refraction was ordered full and in three weeks the squint now measured as follows:

WITHOUT GLASSES

Perimeter	21°
Cover	20°
Synoptophore	19°

WITH GLASSES

Perimeter	18°
Cover	20°
Synoptophore	18°

Atropine was again used three months later with essentially the same findings as already listed.

The left eye was then operated upon, a recession and resection being performed. The

question of a divergence insufficiency, as compared to a convergence excess, was considered but the extreme angle of the squint seemed favorable for a recession of the medial rectus in addition to a resection of the lateral rectus.

Orthoptic training was instituted 10 days after surgery and three weeks after surgery the following findings were noted:

WITH GLASSES

Cover	5°
Synoptophore	4°
Perimeter	4°

WITHOUT GLASSES

Cover	8°
Synoptophore	9°
Perimeter	8°

Residual accommodation was measured in each instance that atropine was employed by means of the Prince rule.

Five months after surgery, second-degree fusion was obtained on the synoptophore with no difficulty and, at certain times, even third-degree fusion was evidenced. An atropine refraction was performed at this time and the following data were obtained. Prior to atropine:

WITH GLASSES

Cover	5°
Synoptophore	4°
Perimeter	4°

WITHOUT GLASSES

Cover	8°
Synoptophore	9°
Perimeter	8°

After one-percent atropine, one drop, three times daily for four days:

WITH GLASSES

Cover	9°
Synoptophore	10°
Perimeter	11°

WITHOUT GLASSES

Cover	14°
Synoptophore	15°
Perimeter	15°

Refraction was: O.D., +3.75D. sph. ⌒ −1.5D. cyl. ax. 165°; O.S., +3.75D. sph. ⌒ −1.5D. cyl. ax. 165°.

Visual acuity was: O.D., 6/9+, with correction; O.S., 6/9+, with correction.

One year following surgery an atropine refraction was again performed, the findings were. Prior to atropine:

WITH GLASSES

Cover	5°
Synoptophore (first degree fusion)	4°
Perimeter	5°

WITHOUT GLASSES

Cover	7°
Synoptophore	8°
Perimeter	6°

After one-percent atropine, one drop three times daily for four days:

WITH GLASSES

Cover	8°
Synoptophore	9°
Perimeter	10°

WITHOUT GLASSES

Cover	13°
Synoptophore	10°
Perimeter	13°

Refraction was essentially the same and visual acuity was correctible at this time to 6/8, O.U. When the condition was checked one month later, the findings were found to be practically the same as those shown before administration of atropine.

Examination one year later revealed no significant changes. Orthophoria and second-degree fusion were noted. Third-degree fusion had been obtained during a short period of concentrated orthoptic training. Two years following surgery, an examination gave results similar to those obtained the year before.

CONCLUSION

This case emphasizes the gaps presently existing in our classification of strabismus. It would seem that the dissociation of convergence and accommodation is a central rather than a peripheral manifestation. Further investigation of reactions of this type should be of marked interest.

735 Clinton Avenue (4).

SOCIETY PROCEEDINGS

Edited by DONALD J. LYLE, M.D.

COLLEGE OF PHYSICIANS OF PHILADELPHIA

SECTION ON OPHTHALMOLOGY

March 15, 1951

DR. WILFRED FRY, *chairman*

WOUND CLOSURE WITH CATGUT

DR. F. BRUCE FRALICK, Ann Arbor, Michigan, the guest speaker, gave an account of his experimental work and experience with catgut in ocular surgery. He recommended 6-0 mild chromic catgut for general use in ocular surgery.

Discussion. Dr. Edmund B. Spaeth: By mild chromic does Dr. Fralick mean the 10-day or the 20-day? Are those approximate?

Dr. Bruce Fralick: Yes.

Dr. Edmund B. Spaeth: Would Dr. Fralick tell us how he handles, in so far as suturing is concerned, his complications to cataract surgery, and perhaps including therein how he uses sutures for corneal lacerations not necessarily connected with cataract surgery?

Dr. Bruce Fralick: When you ask about the complications of cataract surgery, you mean?

Dr. Edmund B. Spaeth: Those which need subsequent suturing.

Dr. Bruce Fralick: We use 6-0 mild chromic gut wherever we used to use silk. We use it for suturing conjunctival flaps. Some of the men have used it in suturing the cornea directly. If we have a rupture of the wound subsequently, or a rupture at the limbus, or ruptures in the sclera, we use 6-0 mild chromic gut to close the lacerations in the sclera. We use 6-0 mild gut in lid lacerations for closing the muscle bundles and for closing the laceration of the tarsal plate. In fact, we use silk very little except in the skin at the present time.

Dr. I. S. Tassman: I would like to ask a question, but, before I do, I wonder if I would be permitted a comment on Dr. Fralick's reference to the delayed proliferation of fibroblasts which sometimes takes up to four days before it really starts. We have sections which will show that the use of plasma and thrombin following cataract sections causes proliferation of fibroblasts within 24 hours, as compared with controlled sections in eyes in which the plasma and fibrin and thrombin were not used. I do not know what the effect of plasma and thrombin might be on the catgut sutures, but I do know that it would reënforce and give more immediate sealing of the wound in association with silk sutures.

As Dr. Fralick stated, the silk sutures might not hold tight for as long as six days, but I am sure the wound will be closed after 24 hours with the use of plasma and thrombin solutions.

My question is: In cases like dislocated lens, for example, or when the eye is under pressure, what security do you have during the course of the operation when you might have reason to expect an immediate expulsion of lens or escape of vitreous when the section is made? How much security do you get, and what benefit is obtained during the course of the operation from all of the post-placed sutures? For postoperative security, closure of the wound with a silk suture fortified by the use of plasma and thrombin will provide the desired closure and much earlier wound healing.

Dr. Bruce Fralick: Dr. Tassman has made a very good point, because placing of sutures across the corneal scleral wound is not easy. Those tissues are very tough and passing even the best available needles through the cornea is done with considerable resistance. If the presence of fluid vitreous

is suspected, I always place my sutures beforehand. There are, however, those cases in which vitreous escapes immediately following the section, before the lens has been compressed at all. Personally, I still would like to have the wound sutures, if I possibly could, whether there is fluid vitreous or not. I feel that the chances for a good result with the least amount of astigmatism are better.

In such cases as Dr. Tassman described, it is quite likely that a McLean or Verhoeff suture placed before sectioning would be the ideal procedure. However we cannot always tell beforehand what the complications will be. They happen unexpectedly.

M. Luther Kauffman,
Clerk.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

February 5, 1951

DR. SAMUEL GARTNER, *president*

ESSENTIAL ATROPHY OF THE IRIS AND SECONDARY GLAUCOMA

DR. DANIEL KRAVITZ presented the case of a woman, aged 27 years, who came to his office on January 28, 1950, with a history of progressive loss of vision for over five years. Prior to that time, she had worn thick lenses for nearsightedness so that at 13 years of age, she was placed in a sight conservation class. The patient could not work because of poor vision.

On January 3, 1945, she went to the eye clinic of the Brooklyn Eye and Ear Hospital; at that time vision was light perception in the right eye and fingers at one foot in the left. The tension was four-plus, digital, in both eyes. She was placed on miotics but without any effect so that on August 31, 1946, a trephining operation was performed on the right eye. At no time following operation was there any improvement in tension.

Examination showed that vision was light perception in both eyes. The right pupil was pinpoint in size and displaced nasally to about two mm. from the limbus. Above, there were evidences of a previous operation, the opening of which was completely closed. Just below this area was a small slit in the iris through which the fundus details were clearly visible.

These showed advanced myopic degenerative changes. The eye refracted over 20 diopters of myopia. There was a diffuse atrophy of all the layers of the iris.

The left pupil was also pinpoint and drawn nasally to about two mm. from the limbus. Opposite to the pupil on the temporal side, there was a large horizontal rent in the iris. The rest of the eye showed the changes noted in the right eye. Tension in both eyes was four plus, digital.

The gonioscopic findings were the same for both eyes—adhesions of the iris to the trabecular zone. There was a considerable amount of pigment deposited on the parts of the trabecular zone which still could be visualized.

DFP (0.1 percent) was prescribed to be instilled morning and night. These drops gave the patient a severe pulling in the eyes which lasted for 15 minutes. However, by February 15, 1950, the intraocular pressure had dropped to 45 mm. Hg (Schiøtz) in the right eye and 25 mm. Hg in the left eye. Two weeks later, the pressure rose to 60 mm. Hg in the right eye and to 30 mm. Hg in the left. Carcholin (one percent, every four hours) was then prescribed, with the DFP to be instilled at night only, for the right eye; while the left eye was continued on DFP.

On April 13, 1950, tension in the right eye was 45 mm. Hg; in the left, it rose to 90 mm. Hg. The left eye was then placed on carcholin (one percent, every four hours) with DFP to be instilled nightly, and the tension promptly began to drop.

Subsequently, the tension in the right eye rose and fluctuated between 65 mm. Hg and 45 mm. Hg (Schiøtz) while that in the left remained in the lower 20s.

Since the right eye did not respond to any miotics, a cyclodialysis and an iridencleisis were performed on the temporal side on July 3, 1950. Immediately after the operation, the tension dropped to 20 mm. Hg and remained so for five weeks, and then rose to 60 mm. Hg. DFP (0.05 percent) was then prescribed, one drop morning and night, and the tension promptly dropped to 30 mm. Hg.

The DFP was then cut down to once daily and the tension has remained under 30 mm. Hg to date. The left eye has been continued on carcholin (one percent, every four hours) and DFP at night only, with the tension never going above 23 mm. Hg (Schiøtz).

Optic nerve and chiasm degeneration

Dr. Abraham Schlossman said that the retina, optic nerves, and chiasm were observed in a patient with bilateral cecocentral scotomas and normal-appearing fundi. The area of degeneration corresponded in large part to the papillomacular bundle. The vessels in the choroid, septa of the optic nerves, and chiasm showed sclerotic changes in their walls. No etiology was determined for the selective atrophy of the papillomacular bundle.

The present case was compared to several from the literature and the nature of the initial pathologic process was theoretically discussed.

A central, paracentral, or cecocentral scotoma in the presence of a normal-appearing fundus must be considered as a symptom in a wide variety of diseases and intoxications. Since pathologic material is not available early in a disease, the retina, optic nerves, and chiasm may present similar histologic findings in diseases with very different etiologic factors.

Discussion. Dr. Max Chamlin said that there is really no difference in the fields in retrobulbar neuritis and toxic amblyopia. As to etiology, he said that from the neurologist's point of view all these cases are potential cases of multiple sclerosis or have multiple sclerosis. Involvement of the chiasm gives a scotoma only when there is a mass, not when there is only an inflammation.

Dr. Schlossman said that the papillomacular bundle goes through the chiasm, and is located in the posterior part of the chiasm; therefore, the posterior chiasm gets the bulk of the damage. Scotoma is simply a symptom. It may be due to multiple sclerosis, avitaminosis, or other causes; scotoma is itself not a disease, but rather the symptom of a disease.

Unilateral cyst of the retina

Dr. Edward Saskin said that, if we view this condition as a cystoid retinal degeneration, the disease is a common one; if viewed in the light of an active accumulation of fluid, the condition is a rare one.

Congenitally conceived cysts are usually associated with microphthalmos and colobomas; the cyst may represent the culmination of peripheral retinal cystic degeneration, in which case it appears as a sharply localized globular detachment of the retina; traction cysts may form due to the pull exerted by shrinking vitreous and preretinal fibrous bands; cysts may form in atrophic areas of retinitis or following trauma or uveitis; and then they are parasitic cysts.

Clinically they appear as tumorlike masses or are associated with widespread retinal detachment. The diagnosis depends upon the relatively static size and shape, transillumination, and, if tapped, the fact that a clear fluid is obtained and the cyst collapses.

He presented J. C., a 66-year-old white man, who came to the Brooklyn Eye and Ear Clinic in August, 1950, with a history of a cataract extraction of the right eye, 10 years previously. He now had an immature cataract of the left eye, which obscured fundus detail, and vision of 5/200, unimprovable. The right fundus revealed nothing. A combined intracapsular extraction was performed on the left eye on September 13, 1950. Convalescence was uneventful except for slight superonasal iris prolapse which worsened after discharge from the hospital.

Study of the eye now revealed not only

the prolapse of the iris but also a globular, saccular, dependent mass in the superonasal quadrant of the retina near the periphery. The mass transilluminated clearly and did not change in size and shape.

Shortly thereafter, the prolapsed iris was excised and a conjunctivoplasty was performed, with uneventful recovery. The cyst mass remained unchanged and corrected vision in this eye is 20/40 minus. To date, Dr. Saskin said, we have no intention of tapping the cyst wall unless there is further marked enlargement of the cyst with decreasing acuity.

Discussion. Dr. Kornzweig said that he had had a case of bilateral retinal cysts. However, in his case the cysts, which were at the 6-o'clock position, had the usual characteristics of retinal cysts; they transilluminated clearly and had a clear wall. After 10 years they remained stationary. He asked whether in Dr. Saskin's case there was any cyst in the other eye.

Dr. Saskin replied that no cystic changes or any other type of degeneration was found in the other eye.

Sympathetic ophthalmia with complete recovery

Dr. Benjamin Friedman presented the case of a man, aged 41 years, who was struck in the left eye by a chunk of wood on April 6, 1949. A laceration over the nasal ciliary region was repaired by his local oculist; an iridectomy was performed and the wound was covered with a conjunctival flap.

Three weeks after the accident the patient noted difficulty in reading fine print with the uninjured eye. A diagnosis of sympathetic ophthalmia was made and enucleation of the left eye was advised. The patient consulted a series of ophthalmologists who all gave him the same advice.

Dr. Friedman said that he saw the patient on May 16, 1949. At that time the corrected vision in the right eye was 20/20; in the left eye, 20/50 plus. Both eyes presented posterior corneal deposits, with cells in the anterior chamber. There were Koeppe nodules on the iris of the right eye. Both eyes were white.

The patient was hospitalized and given the usual antibacterial medication consisting of aureomycin, penicillin, and streptomycin. He was also given salicylates, typhoid vaccine, and milk-injections. At the end of 12 days, the patient was discharged with the understanding that he would continue this type of treatment at his home in Canada.

He was not seen again until November 24, 1950. At this time all inflammatory phenomena had disappeared. The vision in the right eye was 20/20; in the left 20/30.

It was felt that this was a mild form of sympathetic ophthalmia, and that recovery would have taken place with or without medication, and that the patient's judgment in refusing to have the injured eye removed turned out to be proper.

Discussion. Dr. Sidney A. Fox said that he had a patient with sympathetic ophthalmia, who had been advised not to have the eye removed. When he saw the case, the vision in the exciting eye was very little, and he advised removal of the eye. In regard to sympathetic ophthalmia in general, Dr. Fox said he felt it wise to remove the exciting eye when there was no useful vision left. He also stated that he had never seen a case such as described by Dr. Friedman.

Dr. Samuel Gartner asked if any tests had been done to confirm the diagnosis.

Dr. Isadore Givner said that sympathetic ophthalmia is usually considered to be a granulomatous uveitis. He asked if there were any cases seen which were not granulomatous uveitis.

Dr. Kornzweig cited the case of a patient who, following a cataract extraction, developed an inflammation which was diagnosed as sympathetic ophthalmia. The sympathizing eye developed glaucoma, and the exciting eye got well and remained well. The eye was removed and did not show sym-

pathetic ophthalmia, but did show a nongranulomatous uveitis with secondary glaucoma.

Dr. Friedman replied that it is impossible to verify the diagnosis of sympathetic ophthalmia unless a pathologic specimen is available. One cannot make a positive diagnosis clinically; however, in the case presented, the diagnosis is reasonable in view of the history. The patient was not subjected to the uveal pigment test to verify the diagnosis.

Dr. Friedman also said that he did not believe that there were any nongranulomatous cases of sympathetic ophthalmia. He also recalled two cases which developed sympathetic ophthalmia following cataract extraction. They both cleared after removal of the exciting eye, with preservation of more than useful vision in the sympathizing eye.

Relief of intractable blepharospasm

Dr. Sidney A. Fox presented a paper on this subject which was published in the American Journal of Ophthalmology, volume 34, page 1351, October, 1951.

Galactose cataract

Dr. Carl Reiter and Dr. Mortimer Lasky presented a paper on this subject (See: *Am. J. Ophth.*, **35**:69 (Jan.) 1952.)

Angioma of the choriod

Dr. Milton Berliner and Dr. Goodwin Breinin presented a paper on this subject. Dr. Breinin said that angioma of the choroid, circumscribed or diffuse, is the basic ocular lesion of the Sturge-Weber syndrome (encephalo-trigeminal angiomatosis). It also occurs as an isolated tumor visible ophthalmoscopically as malignant melanoma. Criteria for differentiation were listed with the suggestion that angiography may be a useful tool in outlining vascular ocular tumors.

A case of Sturge-Weber syndrome with buphthalmos and a case of isolated choroidal angioma appearing as a tumor in the fundus were presented with pathologic reports on the enucleated eyes of each. Basic similarities between the two conditions were discussed and it was concluded that the isolated tumor represents a variant of encephalotrigeminal angiomatosis.

Bernard Kronenberg,
Recording Secretary.

Ophthalmic Miniature

> Blindness not uncommonly occurs in cases of tumor of the brain. Sometimes the optic nerves are compressed, and here the blindness is easily accounted for. Sometimes also one optic nerve only, on the cerebral side of the commissure, is affected, and then we get half-blindness of each eye. In many cases, however, it is somewhat difficult to account for the production of the amaurosis. . . . The blindness in cases of tumor of the brain, not directly affecting the optic tracts, may be produced by the general pressure, or by reflex irritation, or because, in some way, the tumor interferes mechanically with the blood-vessels, so that parts at a distance do not receive their due supply of blood.
>
> John Hughlings Jackson, 1863.

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700 North Michigan Avenue, Chicago 11

LAWRENCE T. POST, *Consulting Editor*
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IV PAN-AMERICAN CONGRESS OF OPHTHALMOLOGY

At the registration desk on Sunday, January 6, at the Hotel del Prado in Mexico City, the members and guests of the Pan-American Association of Ophthalmology were each handed a large envelope containing a handsome enameled metal badge, a program, a book of abstracts of the papers to be presented, and a stack of invitations by our generous hosts for brilliant and exciting social events for the ensuing week.

From this moment on, time (packed with pleasure and scientific information) flew by until the closing ceremonies on Saturday, January 12. No matter if there was little allotted for sleep, that could be caught up later. Each moment was seized and relished, and each day had more than 24 hours.

The opening ceremonies, held in the assembly hall of the beautiful Palace of Fine Arts, were inaugurated by the secretary to the president of the Mexican Republic, señor licenciado Rogerio de la Selva. Short addresses were made by the president of the association, Conrad Berens; the general secretary

of the congress, Luis Sánchez Bulnes; the president of the congress, Antonio Torres Estrada, and messages from the Portuguese-speaking delegates by Moacyr E. Alvaro, from the French-speaking delegates by Henri Pichette, from the English-speaking delegates by Bernard Samuels, and from the Spanish-speaking delegates by Jorge Valdeavellano.

Interspersed were musical selections by the orchestra of Maestro Jesús Martínez and lovely songs by the eminent soprano, Amparo Guerra Margám.

At the conclusion of the inaugural ceremonies, busses were provided by our thoughtful hosts and we were carried through the beautiful sunlit streets of Mexico City to the Rancho del Charro where we witnessed a "jaripeo," or Mexican cowboy festival of expert riding, roping, bull-throwing, and native dances. This was followed by a delicious "paella," a Spanish dish of rice, chicken, and sea food, washed down with ice-cooled beer and tequila.

We were taken then by busses to the Plaza de Toros Mexico, where many of us witnessed our first bull fight, some with mixed emotions. Eight powerful, black, sleek toros were skilfully despatched by Antonio Velazques, Jesús Cordoba, both of Mexico, and José Maria Martovell of Córdoba, Spain. The drama, the excited crowd, the color and bright sunshine, and the shouts of "Ollé" will not soon be forgotten.

The next morning the scientific sessions began in earnest and continued through the 11th of January. Each hour was devoted to ophthalmology. Twelve symposia, 25 plenary-session papers with formal discussions, about 100 free papers, a round-table on cataract surgery, a plenary session on the prevention of blindness, and one on experimental research in ophthalmology were presented.

The Mexican Society for Ophthalmology, whose president is Abelardo Zertuche, held a meeting on January 8th and scientific addresses were given by the distinguished foreign guests of the congress, Sir Stewart Duke-Elder, Manuel Márquez, Rudolph Thiel, and Edmond Velter. At its conclusion, diplomas were awarded to a large number of newly elected honorary and corresponding members of the Mexican Society for Ophthalmology.

In addition to the scientific programs, 28 films of ophthalmic interest were shown during the meetings, and a fine scientific exhibit of 34 displays received much attention. Commercial exhibits of books, instruments, apparatus, and equipment were likewise well attended.

The second Harry S. Gradle Lecture was given by A. Urrets Zavalía, Sr., whose subject was "Teaching of ophthalmology in the Spanish-American countries." The Pan-American Gold Medal of the National Society for the Prevention of Blindness of the United States was conferred on Alberto Vásquez Barrière of Montevideo, by William L. Benedict, vice-president of the society. Diplomas of honorary membership in the National Society for the Prevention of Blindness were awarded to Señora Adela Formoso de Obregón Santacilia and to Luis Sánchez Bulnes by Franklin M. Foote, the society's executive director.

Social events, besides many lovely private parties, included a cocktail party by the Secretary of Foreign Relations of Mexico, a concert of Mexican folk dances and music, and a brilliant closing ball on the evening of the 11th. The folk dancers, in fresh, bright costumes of various regions of Mexico, formed an extraordinary spectacle of bright primary colors, eagle feathers, plumes of various kinds, and intricate, lovely dance figures. The climax scene of Aztec human sacrifice is unforgettable in its beauty of presentation. The ball, held at the Jockey Club, was attended by everyone connected with the congress. Dance music, ballad singing, entertainers, and beautiful women gave a continuous kaleidoscope of sound and color until early in the morning.

Each day during the congress, E. R.

Squibb and Sons of Mexico published a most entertaining newspaper, or diary, pertaining to the meeting. Profusely illustrated with pictures of many of the actors in the events, amusing caricatures of persons of prominence, historical illustrations and articles, running comments of events of the congress, and a daily editorial of interest, it succeeded in capturing the gaiety and pleasure as well as the importance of the scientific presentations. The six issues of the "Diary" will be a cherished souvenir to all of us, even if some cannot read Spanish fluently.

Tribute should be paid here to our Mexican colleagues who worked so hard to make the congress the success it was, and to their open generosity and hospitality to us. We are particularly grateful to the members of the local organizing committee, Torres Estrada, president; Puig Solanes, vice-president; Sánchez Bulnes, general secretary, and to the various secretaries, Palomino Dena, Chavira, Agundis, Espino, Silva, Hinojosa, Zertuche, Cherif, and Ramviez.

Edward Hartmann, Count Arruga, Manuel Márquez, and Sir Stewart Duke-Elder were elected honorary members of the Pan-American Association. Mrs. Harry S. Gradle was also elected an honorary member and given the title of Executive Director of Prevention of Blindness activities.

The following officers of the association were elected: President, Dr. Moacyr E. Alvaro; executive secretary for South America, Dr. Jorge Balza; executive secretary for North America, Dr. Thomas D. Allen; assistant secretaries, Dr. Brittain F. Payne, Dr. Daniel Snydacker, Dr. Jack Matthews, Dr. A. Edward Maumenee, Dr. Alfred Elliott, Dr. J. Wesley McKinney, Dr. McKay, Dr. Palomino Dena (Mexico), Dr. Ricardo Fernandez (Puerto Rico), Dr. Oscar Horstman (Cuba), Dr. Rene Contardo (Chile), Dr. Alcides del Ciello (Brazil), Dr. Renato de Toledo (Brazil), and Dr. Alfonso Gaitan Nieto (Colombia).

Vice-presidents of the association are: Argentina, Dr. Esteban Adrogué; Bolivia, Dr. Aniceto Solares; Brazil, Dr. Hilton Rocha; Canada, Dr. Henri Pichette; Chile, Dr. Santiago Barrenechea; Colombia, Dr. Francisco Vernaza; Costa Rica, Dr. Alexis Aguero; Cuba, Dr. Miguel Branly; Ecuador, Dr. J. M. Varas Samaniego; El Salvador, Dr. Humberto Escapini; Guatemala, Dr. R. Pacheco Luna; Haiti, Dr. Elie Villard; Mexico, Dr. Raul A. Chavira; Nicaragua, Dr. Ricardo Lacayo; Paraguay, Dr. Honorio Campuzano; Peru, Dr. Jorge Valdeavellano; United States, Dr. William L. Benedict and Dr. Derrick Vail; Uruguay, Dr. Raul Rodriguez Barrios; Venezuela, Dr. J. M. Espino.

The V Pan-American Congress will be held the first week in February, 1956, at Santiago de Chile.

At the conclusion of the executive meeting, a small group of officers and members proceeded to the statue of Dr. Camona y Valle adjacent to the medical school where a memorial wreath was placed following appropriate remarks by Alvaro, Berens, and Duke-Elder, who pointed out the important part Camona y Valle played in the establishment of the first free eye clinic in Mexico City and his great influence on Mexican ophthalmology.

Derrick Vail.

SPECIALIST EXAMINATIONS IN CANADA

Readers of the JOURNAL no doubt will be interested in the story of the development in Canada of qualifying examinations in ophthalmology—of what has come to be called "certification," as Canadians in designing their examinations have been influenced greatly by the American Board.

Ever since the days of confederation the licensing of medical practitioners in Canada has come under provincial jurisdiction. Since each province has been jealous of its prerogatives, the difficulties of superimposing a nationwide specialist-qualifying examination can be well understood. Nonetheless for

many years a widefelt need has existed in Canada for examinations indicating special qualifications.

At first this need was felt most keenly in respect to general medicine and surgery. To meet it, young medical graduates frequently would take one or other of the "Fellowship" examinations in Great Britain. In ophthalmology, no particular qualification was required, but many took the D.O.M.S. (England) or the American Board. With the growth of the profession not only in size and complexity, but perhaps more importantly in national pride, the need for a truly Canadian system was recognized.

In 1929, through the agency of the Canadian Medical Association, a bill was passed in the Canadian parliament, and given royal assent, for the formation of the Royal College of Physicians and Surgeons of Canada. This body since has held scientific and business sessions, and it has held yearly examinations for the higher qualification of physicians and surgeons.

In 1939, the college was given the right by parliament to issue certificates of qualification in several specialties. As very few ophthalmologists in Canada held a fellowship in the college, immediately the question arose as to who would be the examiners. As often occurs in such cases there was much jockeying for position among various interested parties. The Canadian Ophthalmological Society, though the representative body in Canada, did not feel that it was its responsibility to examine medical men.

The issue was forced in 1943, when the provinces of Alberta and Ontario instituted legislation requiring specialists to be qualified. After prolonged discussions, an arrangement was worked out by which the Canadian Ophthalmological Society nominated an examination committee which then was appointed by the Royal College.

In practice it has become the rule that the heads of the departments of ophthalmology of the various medical schools form this committee. Up until 1944, certification of properly qualified individuals was permitted without examination. But since that date certification essentially has been by examination only. Up to 1951, 312 have been certified without examination and 46 have been certified by examination.

The conditions for admission to examination are somewhat stricter than those for the American Board. It is required that the candidate have:

1. Satisfactory moral and ethical standing.
2. Graduation from a medical school approved by council.
3. The candidate shall produce documentary evidence of supervised study and/or practice during an aggregate period of at least five years after graduation: (a) One year shall be spent in a general interneship approved by council; (b) two years shall be spent in study and training in the specialty in a hospital or hospitals organized for training in this specialty (A period of the training shall be spent in the study of internal medicine or general surgery, and the basic medical sciences necessary to the proper understanding of the specialty.); (c) study and/or practice of the specialty for the remaining portion of the five-year period.
4. If the training has been interrupted or taken on a part-time basis, the training of the candidate must be satisfactory to council.

In the examinations an attempt is made to combine the best features of those given in England and the United States. They are made up of a written part consisting of two papers, one on the basic sciences and one on the clinical aspects, and of an oral part.

It always has been the hope that arrangements could be worked out for the reciprocal recognition of these examinations with the American Board. Since the latter is a basic requirement for admission to many American ophthalmic societies, and as so many Canadians wish to join these bodies, such reciprocity would be most helpful. But these hopes have failed to materialize. This is one of the severest criticisms in Canada of present arrangements.

Another is that, at the present moment, the young ophthalmologist spends so many of his most active years jumping the hurdles that he has little energy left for creative thinking and work. In Canada today, a young medical man must take his usual university examinations and his provincial licensing examinations. If he has any ambition, and sometimes from necessity, he must obtain the license of the Medical Council of Canada. Having completed his postgraduate training, he then takes the certification examination of the Royal College. In most provinces he must have this qualification before he may practice his specialty, or even call himself a specialist. In some provinces an equivalent qualification will be accepted. In other provinces, such as Quebec, there are severe penalties if he does not comply. Then if he wishes to join the more important American ophthalmic societies he must pass the American Board.

Recently, another rung has been added to the ladder. Now some medical schools are demanding even a higher qualification before admission to their teaching staffs. This is the Fellowship of the Royal College of Surgeons of Canada in Ophthalmology, which was instituted in 1948. The requirements for admission to examination are five years' study, with one year in surgery, metabolic diseases, or neurosurgery. Forty percent of the examination is in general medicine and surgery as related to ophthalmology. The examination board consists of two or more ophthalmologists, and at least one physician and one surgeon. Insofar as qualification goes the "fellowship" is accepted in lieu of certification. Though this is true theoretically, in practice usually both examinations are taken.

It is not surprising that many are worried by the possible effects of all this regimentation. It is admitted by all that some form of basic qualification is desirable. But it is not admitted by all that medicine in its highest form can be assured by such procedures.

On the contrary, many feel that the burden of examinations, and the resulting regimentation of thought and effort will be a depressant. They feel the product will be a conventionalized caricature of what a progressive medical man should be. They believe that those who control the destiny of the profession have lost sight of the fact that the practice of medicine at its best requires not only a well-informed mind but a creative one, and that it is largely an art based on love and compassion for mankind. It is stated by others, equally vehemently, that these fears are baseless. It is possibly significant that the majority of those who make the rules have not passed through the examination mill.

What the ultimate result may be will take a considerable time to determine. For the moment at least, the pendulum has swung well over in the direction of control.

John V. V. Nicholls.

CORRESPONDENCE

Ocular manifestations of some tropical diseases*

Editor,
American Journal of Ophthalmology:

I should like to compliment Dr. Samaniego-Varas, Guayaquil, Ecuador, on the excellence of his paper. I am certain it has been most stimulating and interesting to all of us. The records of his extensive clinical experience in tropical ophthalmology are most illuminating.

Few eye diseases are found exclusively in the tropics. Certain other eye conditions, although occurring in many parts of the world, are much more frequently encountered in the tropical zone. The acute systemic reaction and the general debility of the malarial patient is well known and fully appreciated by the physician of the subtropical and tropical regions.

* Through oversight, this discussion was not published as intended with the article, "Ocular manifestations of some tropical diseases," by J. M. Varas Samaniego, appearing in the Journal, volume 34, page 1574, November, 1951.

Estimation of the frequency of eye changes in malaria varies from rare to 10 percent. Many ocular complications have been described and some changes are open to question. Elliot and others from their extensive experience in India were inclined to doubt a high incidence of complications. It has been suggested that different types of plasmodia may be responsible for the variations in recorded literature.

An accurate estimation of ocular complications in malaria is not easy. It is uncommon for an ophthalmologist to see a large number of acute malarial cases and it is probably just as seldom that the physician who treats malaria practices daily routine ophthalmoscopy. For this reason it is clear that many minor ocular complications could easily escape detection.

The experience gained in treating malaria during World War II was considerable and the systematic recording of this material may help to reëvaluate former ideas.

I should like to summarize my experience with the ocular manifestations of malaria at Gorgas Hospital in the Panama Canal Zone during four years of the war. Malaria was especially prevalent during the early phases of the conflict when so many troops were occupying poorly sanitated jungle positions. From the years 1942 through 1945 there were 8,068 cases of malaria admitted and diagnosed at the hospital.

The conjunctivas of malarial cases often showed a moderate injection during the acute febrile state such as may happen in any fever. Certain severe cases showed a yellowish appearance of the conjunctiva which is said to be associated with the hemolytic effect of the parasitized red cells.

The usual corneal lesion associated with malaria was dendritic keratitis. This condition occurred in only seven instances during the acute attack of malaria. It was discovered usually between the first and third week of the disease. Chamberlain and Bronson, in writing of their experience with troops in the tropics, stated that dendritic keratitis occurred in one for each 188 new malarial cases, or six times more frequent among troops infected with malaria. Other Army surgeons have reported that the incidence of dendritic keratitis was no higher in malarial patients than in nonmalarial patients.

In addition to the acute cases we also observed a dendritic keratitis in eight cases of chronic malaria in which the patient was fever-free and malaria was wholly unsuspected. These eight cases were admitted directly to the eye service since a painful eye was the sole complaint. The malaria, both tertian and estivo-autumnal, was discovered two to nine days following admission. In no case was malaria suspected by the admitting officer since temperatures were usually normal and never more than 99°F.

Careful study of thick and thin blood smears over a period of several days was necessary before a positive identification of the plasmodium was possible. In two instances it was found necessary to resort to a method to "coax out" the plasmodium into the peripheral blood.

As a provocative in the diagnosis of malaria, adrenalin and nicotinic acid were used; 200 mg. of nicotinic acid were given and followed in 30 minutes by three minims of adrenalin. Routine thick and thin smears were taken at the time of the adrenalin injection and then at one and two hours after the injection. This method was found effective in demonstrating malaria in the blood smear when the ordinary methods failed.

The acute attack of malaria had been from six months to a year prior to the keratitis. The keratitides healed in all cases following treatment of the malaria and local treatment to the eye. There were no recurrences.

Evidence has accumulated which indicates that dendritic keratitis is probably due to a virus. However, in our series, attempted transference of scrapings from a dendritic ulcer to a healthy cornea was unsuccessful in two cases. No other form of keratitis was

observed in association with malaria—either of the acute or chronic form.

One case of dendritic ulcer was so refractory to treatment that it appeared to develop into a localized deep form of keratitis which eventually healed. No interstitial keratitis or keratitis profunda from malaria was observed.

Small retinal hemorrhages in malaria were seen very rarely. These fundi were classified as septic retinitides, for the scattered small hemorrhages were in no way typical of any disease but resembled what may be found in certain acute infectious diseases. The hemorrhages disappeared promptly, leaving no trace of their former existence.

No large preretinal or subhyaloid hemorrhages were seen in malaria cases. It has been my experience that these hemorrhages are nearly always associated with a profound anemia and low platelet count. It is likely that their occurrence in malaria would be as a result of the anemia that sometimes accompanies the disease rather than the malaria itself.

Malarial optic neuritis occurred in two cases. They occurred during the second week of the disease with a sudden onset of bilateral loss of central vision approximately equal in the two eyes. The nerveheads appeared typical of a neuritis, with swollen, hyperemic discs. Treatment of the malaria was continued with atabrine, and large doses of thiamine chloride were administered. Vision gradually returned to normal during the next six weeks but there was a mild residual pallor of both discs.

There were no cases of iritis, uveitis, paralyses of the extraocular muscles, or complete amaurosis which have been described in association with malaria. During convalescence many patients complained of an asthenopia especially for close work but this symptom disappeared when their general condition returned to normal.

One of the most distressing conditions for the internist and ophthalmologist alike was the occurrence of quinine amblyopia. It varied from complete amaurosis to mild constriction of the visual fields. Individual susceptibility to quinine was apparent, for in several cases amaurosis developed after ingestion of as little as 40 gr. of the drug.

The ophthalmoscopic appearance of the severe form of quinine amblyopia is characteristic. The pupils are widely dilated, the retina is edematous, and the arterioles are markedly constricted. As recovery occurs, the retinal edema lessens but mild pallor of the disc can be observed.

The vascular constriction usually remains about the same in the severe cases. In the mild cases of quinine amblyopia where there was but slight constriction of the peripheral field, recovery was complete but, in the severe forms, residual pallor and some loss of peripheral field were noted despite the most energetic treatment with vasodilators and paracentesis.

At the first suggestion of amblyopia, quinine was stopped and further therapy was continued with atabrine. Visual recovery was slow, with return of partial central vision first, followed by gradual enlargement of the peripheral field.

Quinine amblyopia must be distinguished from malarial amblyopia. There should be little difficulty if one recalls the difference in the ophthalmoscopic appearance and the characteristic field defects in toxic neuritis versus quinine amblyopia.

There is another condition which should enter the discussion briefly because of its very unusual features. Ocular filariasis in the form of onchocerciasis occurs in this continent in two well-localized areas, the Pacific slopes of Guatemala and southern Mexico. In 1944, I had the rare opportunity of visiting an onchocercotic region as the guest of the Pan-American Sanitary Bureau.

The disease is found especially among the laborers of the coffee fincas at an altitude of 3,000 to 4,000 feet. It is characterized by nodular swellings about bony prominences chiefly the pelvic girdle and scalp. The nodules contain a large female and smaller

male worm coiled together. Thousands of microfilaria issue forth from this union and travel all over the subcutaneous tissue, conjunctiva, and even penetrate the eyeball.

The original filaria are transmitted by the bite of the Simulian fly which breeds in the fast-moving streams of that region. The parasite undergoes part of its development in the fly and part in the human. The fly is reinfected by biting a previously infected human.

The diagnosis is made from small skin biopsies from the ear or conjunctiva where microfilaria can be found in abundance. It has been estimated that, in infected zones, 30 percent of the eyes are involved, although I suspect this figure must vary a great deal.

The presence of microfilaria or their toxins in the eye may cause various changes, depending on the response of the structure involved. The most common corneal lesion I observed was a punctate keratitis. It has been said that these grayish spots are the reactions to microfilaria which have partially penetrated the cornea. However, punctate keratitis does occur without the presence of microfilaria.

The presence of microfilaria in the anterior chamber has been noted under the slitlamp. W. B. Clark in his excellent report on onchocerciasis recovered microfilaria from the aqueous following paracentesis in 29 of 62 patients who gave a positive conjunctival biopsy. Dr. Clark commented on the presence of microfilaria in the aqueous without change in the cornea or iris.

Iridocyclitis and chorioretinitis occur and a choroiditis juxtapapillaris was seen in a few of the onchocercotic cases examined. A few natives whom I saw had dense corneal leukomas which were claimed as the result of onchocerciasis.

Ocular leprosy is not uncommon in Panama nor in the neighboring countries. All known cases are confined to the Leprosarium of the Canal Zone where facilities are at hand for careful study.

In a recent survey, 90 percent of the patients were found to have eye complications; 54 percent of the patients had vision of 6/60 or less and, of this number, 13 percent were blind. In leprosy the anterior segment of the eye was involved almost exclusively.

A typical "chalk-dust" superficial punctate keratitis occurred in 34 percent of all cases. Evidence of iritis occurred in 50 percent of the cases. Pinpoint lepromas of the iris were frequently seen with the slitlamp. Absolute corneal anesthesia was found to be rare but relative loss of corneal sensitivity does occur in severe cases. The leper patient withstood ocular surgery fairly well in the absence of an acute inflammatory process.

Promin was an effective drug in the treatment of leprosy. Secondary infection cleared and recurrent attacks of iritis were definitely less common.

(Signed) Robison D. Harley,
Atlantic City, New Jersey.

Research grants available

Editor,
American Journal of Ophthalmology:

The National Institute of Neurological Diseases and Blindness, under Dr. Pearce Bailey, director, now has funds available for the support of research grants-in-aid, research fellowships, and traineeships in rehabilitation.

Research grants-in-aid are awarded to nonfederal institutions for the support of research projects submitted by qualified investigators in their particular fields of interest.

Research fellowships will be awarded to promising students and scholars for their support in their predoctoral and postdoctoral research training.

In addition, the institute has limited funds for the training of various professional personnel in methods of rehabilitation, in order that they can later assume a teaching function and become the nuclei of rehabilitation groups throughout the country.

Requests for further information and for

grants and fellowship applications should be addressed to Dr. Frederick L. Stone, Chief of Extramural Programs, National Institute of Neurological Diseases and Blindness, Bethesda 14, Maryland.

(Signed) Dan M. Gordon,
New York.

BOOK REVIEWS

GENETICS IN OPHTHALMOLOGY. By Arnold Sorsby, Research Professor in Ophthalmology, Royal College of Surgeons and Royal Eye Hospital; Surgeon, Royal Eye Hospital, London. London, England, Butterworth and Company (publishers) Ltd.; St. Louis, The C. V. Mosby Company, 1951. Price: $9.50.

There is available at last, and in English, a most satisfactory text for ophthalmological genetics! Sorsby's book may be compared most favorably with such classic continental works as Waardenburg's *Das menschliche Auge und seine Erbanlagen*, and/or Franceschetti's contribution in the *Kurzes Handbuch der Ophthalmologie*.

Before discussing the contents, a few words could be said on the general arrangement and appearance of the book. The binding is conservative and the size most convenient for comfortable reading. The print is good and relatively easy on the eyes. The paper is semigloss and takes very well the 233 figures which include instructive and interesting illustrations of ocular disease and family pedigrees. A valuable bibliography of carefully selected references and a comprehensive index are included. The text is arranged in a brief yet concise manner into three major sections: (1) Theoretical considerations, (2) isolated ocular anomalies, and (3) generalized disorders with ocular aspects.

Section I, "Theoretical considerations," presents in an understandable yet most comprehensive manner the principles of modern genetics. The ophthalmologist is not required to shell peas or count corn kernels in order to grasp the basic fundamentals of the science of heredity. Rather the basic knowledge in the field of genetics is treated as it applies to ophthalmology and utilizes familiar ophthalmic material. Sorsby, with seasoned practicality, emphasizes that inherited diseases may be treated and controlled.

Section II, "Isolated ocular anomalies," is presented in seven chapters. Chapter 1, "The globe as a whole," includes a section devoted to the problem of glaucoma and its hereditary aspect. The specificity of hydrophthalmos and glaucoma is supported on a genetic basis. In discussing the "Aberrations of refraction" the author denounces the fallacious attempt "to seek genetic interpretation of refraction and its anomalies by taking into account only the total or clinical refraction of the eye." A true "genetic analysis" will be accomplished only when the "mode of inheritance" and course of development of the variable "components of refraction are known."

The role of heredity in muscle imbalance is outlined for the serious deliberation of the oculist.

Short but adequate descriptions of the known modes of inheritance of the developmental, congenital, and abiotrophic anomalies of the cornea, lens, uveal tract, choroid, retina, optic nerve, lids, and lacrimal apparatus appear in sequence.

Section III, "Generalized disorders with ocular aspects," is an extremely valuable division of the book. The ocular changes associated with hereditary metabolic and systemic diseases and the hereditary syndromes are clearly described and, when possible, adequately classified. The very difficult subject of the syndromes is ably presented and will provide the alert ophthalmologist with a valuable source of diagnostic knowledge.

For the student and the busy practitioner this book is a must!

Harold F. Falls.

OPHTHALMOLOGY. By Arno E. Town. Philadelphia, Lea and Febiger, 1951. Clothbound, 487 pages and index, 208 illustra-

tions and four colored plates. Price: $10.00.

This new textbook was prepared under the able direction of Dr. Town with Dr. Clark, Dr. Duggan, Dr. Fox, Dr. Givner, Dr. Howland, Dr. Kasper, Dr. Leopold, Dr. Naidoff, Dr. Shipman, Dr. Wagman, and Dr. Waldman as contributors.

It is intended for use by undergraduate medical students and general practitioners. For the average undergraduate student it is a somewhat more comprehensive and complex exposition than his actual needs require or the limitation in time in a crowded medical curriculum makes practicable. As a reference work rather than a text for assigned reading it seems to be ideal. For the general practitioner it is likewise adequate for the same purpose; for the postgraduate student in ophthalmology it is sufficiently complete to serve as a means of orientation in the specialty at the beginning of his training and before he begins more extensive reading in more elaborate treatises.

No exception can be taken to the factual material presented nor in the manner of presenting it. The book presents the subject in its most modern sense and, since it is a new book, it carries over no antiquated ideas from previous editions and takes full cognizance of the newer developments in the field. Some 73 pages are devoted to the physiology of vision. The arrangement is logical in that anatomy is not carried as a separate chapter but the anatomic characteristics of each subdivision are considered at the beginning of the chapter devoted to discussion of the disease of that portion of the eye.

For the avowed purpose of this text, the chapters on neuro-ophthalmology, the ocular changes in general disease, and the standing orders for traumatic cases in industrial dispensaries are particularly valuable. It is questionable whether, in a work of this scope, 37 pages should be devoted to surgery of the eye.

The book is adequately illustrated, although one might wish that more color plates had been included, and this will doubtless be done in future editions.

William A. Mann.

Chronology of Ophthalmic Development. By Arthur H. Keeney, M.D. Springfield, Illinois, Charles C Thomas, 1951. Price: $2.00.

This monograph is divided into two main sections. The first section deals with prenatal and postpartum development. It is all told in table form. Each section of the eye is carried through a prearranged time schedule, so it is easy to follow any one type of tissue through all the stages from the third fetal week to 25 years of age; or at any one stage in development one can determine how every tissue of the eye is developing.

The second section deals with the functional development of vision from the third fetal month up to nine years of age.

The monograph, as stated by the author, contains nothing new so far as embryology is concerned. It is a cleverly arranged, brief outline of the subject. It will make an excellent reference for the student, or the practicing ophthalmologist, pediatrician, or general practitioner who has an understanding of embryology. As the author states, "It is a comprehensive review and reference table, not a textbook of the subject."

There is a very definite need for an outline of this type. Nothing similar to it is now in print.

Earl H. Merz.

Bulletin de la Société Belge d'Ophtalmologie, 1950, volume 96, pp. 527-721; 1951, volume 97, pp. 1-217.

Twenty-five papers, which represented the current status of various topics, were read during the scientific session on November 26, 1950.

L. Hambresin and J. Bernolet discussed the ocular complications of smallpox vaccination. They distinguish three types of corneal involvement—a mild marginal infiltration,

the real vaccinia pustule of the cornea, and a disciform type of keratitis which is often accompanied by paralysis of the fourth and eighth nerve. The latter represents an endogenous effect of the virus and belongs to the postvaccinal syndrome of meningo-encephalo-myelitis.

R. Weekers and E. Prijot report on their interesting work on the vasomotility of aqueous veins. Aqueous veins and laminated veins of rabbits contracted immediately when touched with an applicator, dipped in 0.1-percent solution of adrenalin, and the opening up of a second layer of aqueous veins followed. Instillation of a solution of 0.1-percent adrenalin caused a rise of the ocular tension because of an increased resistance to the outflow of the aqueous.

J. Zanen and G. Rausin observed a boy with bilateral cyst formation in the macula, probably congenital, and unusually good visual acuity—0.80 in the right eye and 0.50 in the left. A. Fritz describes his new method of measuring the retinal capillary pressure, using a special strong ophthalmoscopic light and registering the width of the capillaries on the disc by direct inspection, a method which was criticized by R. Weekers because the capillaries of the disc are not identical with those of the retina.

Phototraumatism and the formation of a bilateral posttraumatic cyst of the macula was the topic of Alaert's and Bingen's paper. An anatomico-pathologic study on two unusual types of nevus of the bulbar conjunctiva was made by J. Zanen and Brihaye van Geertrurden. One growth was an intra-epithelial nevus, related to the junctional nevus. The other was a mixed nevus, equivalent to the compound nevus of the skin described by Allen.

An excellent paper on angioid streaks and pseudoxanthoma elasticum and their relationship to vascular diseases and Paget's disease was read by P. Malhiew and J. Dedoyard. They included six case histories.

A. Hoorens and A. Philipps contributed a survey on the pharmacodynamics of miotics and mydriatics. They concluded that the persistent mydriasis after instillation of atropine and miosis after instillation of eserine were caused by fixation of the drug in the tissue of the iris and not by retention in the aqueous.

Appelmans and Forez believe that a one-percent solution of benzedrine is more exact and less harmful than cocaine in checking the function of the cervical sympathetic chain.

H. Moutinho reviewed his experiences with cortisone; J. François reports two cases of intraocular foreign body, found on gonioscopy; R. Weekers and E. Prijot discuss the symptomatology, pathogenesis, and treatment of hypertensive leukemic uveitis.

L. Debals describes the case history of an exudative chorioretinitis and optic neuritis which followed a dental infection by direct extension to the maxilla, orbit, and finally the optic nerve.

A. Fritz studied the retinal circulation in a case of exophthalmic goiter. He calls attention to the high pressure in the retinal capillaries and to its possible relation in the development of exophthalmos.

Appelmans, J. Michiels, and J. De Niel treated a bilateral case of angiomatosis retinae with diathermy and emphasize the importance of retinal exudation in the prognosis of this disease.

J. François discussed the signs, symptoms, and differential diagnosis of scleromalacia perforans, and described a case of his own. The treatment is unsatisfactory and consists in curetting of the sclera, irradiation with ultraviolet light, and cauterization with zinc sulfate. He quotes Paufique who had successfully transplanted a scleral graft, a treatment which should be kept in mind for this disease. François and Rabaey found interesting details in the lens and the lens epithelium with their microscopy "par contraste de phase" and they praise this new method of investigation.

M. Wibo spoke on noncancerous growths of the choroid and retina and their treatment. He suggests Bémagnol which is a combination of magnesium chloride and vitamin B in

tablet form. He cites three case histories of intraocular growth with retinal detachment which were treated, apparently successfully, with Bémagnol, six tablets a day.

The February, 1951, volume contains a series of papers on exophthalmos of endocrine origin. The papers were read as independent lectures and not as parts of a symposium but nevertheless offer a wealth of coördinated and unified information.

Considerable space is given to the principal presentation made by P. Danis and J. Mahaux, "The simple and the edematous exophthalmos of endocrine origin." This monograph includes chapters on the history, pathologic anatomy, and physiology of the orbital and bulbar signs and symptoms, as well as the accompanying disturbances of the blood, fluid, and electrolyte balance and anomalies of the nervous system. Much time was devoted to studies on animals. The experiments show two mechanisms in the development of exophthalmos.

Pharmacologic and electric stimulation of Müller's orbital muscle advances the eyeball to a certain extent. Injection of pituitary extract causes a swelling of the orbital tissue and anterior displacement of the eyeball. The exophthalmos caused by stimulation of the sympathetic nerve occurs without organic variations. It hardly can be noticed in man because of the underdevelopment of the orbital muscle.

The exophthalmos of pituitary origin produces characteristic organic deformities. It becomes apparent also in absence of Müller's muscle, or destruction of the sympathetic and does not disappear after death. There are typical changes of the retrobulbar tissue, edematous infiltration, degeneration and fatty infiltration of the ocular muscles and a pseudoinflammatory infiltration with lymphocytes—findings which correspond completely with the findings after surgical and chemical thyroidectomy in man. Data on the possible prevention and treatment of endocrine exophthalmos are summarized.

M. A. Dollfuss and H. Welti describe the history of two patients with malignant exophthalmos who were improved by decompression of the orbit through the temporal fossa and removal of the lateral and superior wall as described by Welti. The decompression favors the resorption of the edema but, in the presence of a hyperthyroidism, a secondary, partial thyroidectomy should be added.

R. Weekers and J. Dedoyard define the terminology, symptomatology, and pathogenesis of thyrotoxic and thyrotropic exophthalmos and the chances of an adequate treatment.

J. François finds the unilateral retraction of the upper lid, even without proptosis, very suggestive of Graves' disease and of differential diagnostic value in bilateral exophthalmos. He localizes the lesion responsible for the retraction of the upper lid in the diencephalon and mesencephalon whose irritation by the thyrotropic hormone causes the hypertension of the levator palpebras.

A. Fritz studied the effect of cortisone on the retinal capillaries by using his own direct ophthalmoscopy with a specially adapted light. He found that cortisone neutralized the effect of eserine on the retinal capillaries, abolished the hypertensive action of galvanic or faradic stimulation to the cervical sympathetic nerve, and neutralized the hypertension in the retinal capillaries, normally following a retrobulbar injection of tetraethyl-ammonium. This parasympatholytic and sympatholytic action and the possible stabilization and lowering of the capillary pressure seem to be closely connected with the blocking action of cortisone in inflammatory and allergic reactions.

N. Appelmans and J. De Niel compare successes and failure of X-ray therapy in pituitary adenoma.

J. François recommends parenteral estrogens in large doses given for a prolonged period as the treatment of choice in thyrotropic exophthalmos because of the restraining qualities of these drugs on the secretion of the pituitary.

M. Moers discusses a typical case of hypertelorism and its etiology and differential diagnosis.

Alice R. Deutsch.

TRANSACTIONS OF THE 5TH CONGRESS OF THE OPHTHALMOLOGICAL SOCIETY OF TUSCANY, UMBRIA AND EMILIA. (L'Arcispedale S. Anna di Ferrara 4:1-156, 1951.)

This congress took place on October 8, 1950, in the new eye institute of the St. Anna Hospital in Ferrara. This new institute was donated by M. Ascoli (New York). It is equipped with all modern facilities and has 57 beds in 15 rooms, some of which are completely sound-proof.

Among the experimental work reported was a study on the oxygen consumption of the lens by C. Pentini. He studied with the apparatus of Warburg lenses in an enriched glucose (one percent) Ringer-phosphate solution. The lenses were of his animals previously treated with ACTH, desoxycorticosteronacetate, or both drugs. The higher glucose concentration was used because it was previously found that after stress the glucose content of the aqueous increased. In this solution the lens of untreated animals had a higher oxygen consumption than normal in 75 percent of the animals. In the treated animals, there was not only no increased oxygen consumption but an inhibition of the oxygen uptake, especially in the doca-treated animals.

In experiments reported by G. Gualdi, hyaluronidase, injected intravenously, causes a slight increase of the ocular tension and a small degree of miosis.

The influence of short waves on the eye was discussed by M. Simonelli and V. Rizzini. With a wave-length of 3.0 cm., they produced corneal lesions in rabbits and with a wave-length of 10 cm., cataractous changes in the lens.

G. Cucco found an increased permeability of the blood aqueous barrier after the injection of histamine in rabbits. He used the fluorescein method of Amsler. Lysozyme was found to destroy bacillus subtilis in vitro by F. Tallei. He also found that paracentesis alleviated a subtilis endophthalmitis. This was ascribed to the higher lysozyme content of the secondary aqueous.

A. Musini investigated the possible influence of the blood groups and the factor Rh on the tissue therapy with fresh placenta. No such influence could be found and these factors apparently play no role in the expulsion of the transplant.

Among the clinical papers and case reports was a paper read by M. Corrado on the occurrence of central vein thrombosis in the course of a simple chronic glaucoma. Two cases were reported and the author assumed that some change in the chemical composition of the blood was the cause of the thrombosis. E. Magni used Kestenbaum's method of counting the capillaries of the optic disc for evaluating optic atrophy of various origins and got valuable information by this method.

Three cases of brucellosis with ocular manifestations were presented by N. Pagliarani. Two patients had a bilateral uveitis with retinal detachment, a new lead in the search for the etiology of Harada's disease.

L. Nigro examined the records of 30 patients with Eales's disease, in 70 to 80 percent of whom tuberculosis was found. The association of a metastatic uveitis with osteomyelitis was reported by T. Ravalli. The two-year-old child recovered completely after intensive antibiotic treatment.

A case of myasthenia gravis was discussed by G. Rossi. The patient had a ptosis associated with a gaze paralysis for looking upward. A new variation of a ptosis operation was shown in a film by M. Vercella. This seemed to be a combination of the methods of Wheeler and Dickey.

Frederick C. Blodi.

ABSTRACT DEPARTMENT

EDITED BY DR. F. HERBERT HAESSLER

Abstracts are classified under the divisions listed below. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. Anatomy, embryology, and comparative ophthalmology
2. General pathology, bacteriology, immunology
3. Vegetative physiology, biochemistry, pharmacology, toxicology
4. Physiologic optics, refraction, color vision
5. Diagnosis and therapy
6. Ocular motility
7. Conjunctiva, cornea, sclera
8. Uvea, sympathetic disease, aqueous
9. Glaucoma and ocular tension
10. Crystalline lens
11. Retina and vitreous
12. Optic nerve and chiasm
13. Neuro-ophthalmology
14. Eyeball, orbit, sinuses
15. Eyelids, lacrimal apparatus
16. Tumors
17. Injuries
18. Systemic disease and parasites
19. Congenital deformities, heredity
20. Hygiene, sociology, education, and history

2

GENERAL PATHOLOGY, BACTERIOLOGY, IMMUNOLOGY

Fazakas, S. **Ocular mycoses.** Ophthalmologica **121**:249-258, May, 1951.

The offending fungus was identified in every one of the fifty cases of fungus infection of the eye. Six species of fungi not reported previously in the ophthalmologic literature occurred in this series. The distinction between primary and secondary keratomycoses could be made. Most mycoses proved benign and amenable to treatment. Peter C. Kronfeld.

Ferrante, A., and Borio, G. **The behaviour of the pathergic status in ocular pathology.** Arch. di ottal. **55**:23-58, Jan.-Feb., 1951.

The pathergic status, as investigated with the intradermal tuberculin reaction, is of no value in answering the question whether in a given case the disease is of a focal nature or whether the focus is an accidental finding. If one examines the pathergic behaviour before and after removal of a focus in the tonsil and during the clinical evolution of the eye disease, one can frequently observe a transition from "pleoergy" to "pleoesthesy" which might explain the clinical deterioration of diseased eyes with prevalently tuberculous characteristics. John J. Stern.

Hallett, J. W., Leopold, I. H., Vogel, A. W., Cannon, E. J., and Steinmetz, C. C. **Treatment of experimental herpes simplex keratitis in the rabbit.** A.M.A. Arch. Ophth. **46**:33-38, July, 1951.

In the treatment of experimental herpes simplex of the rabbit cornea the authors found that topical instillations of neomycin, aureomycin, orange dye, aureomycin combined with chloramphenicol, terramycin combined with chloramphenicol, and cortisone were of no value. Cortisone acetate suspension injected subconjunctivally increased the intensity of herpes simplex keratitis. Chloramphenicol (chloromycetin) used topically, intensively and early, slowed the progress of the infections, but by the sixth day treated and untreated eyes appeared the same. Topical treatment with terramycin or the antihistamines, when used early and intensively, was beneficial. Tripelennamine (pyribenzamine) hydrochloride administered intravenously before inoculation had a definite protective action. The course of the herpes simplex keratitis

could not be influences when therapy was instituted after the disease had been established. (1 colored plate)

Edward J. Swets.

La Manna, N., and Capolongo, G., **The bacteriology of eyeballs enucleated after perforating injuries.** Arch. di ottal. **55**:59-70, Jan.-Feb., 1951.

Thirteen eyes which were enucleated from 7 days to 50 years after perforating injuries and one which had been removed after a contusion were examined for the presence of infectious organisms by culturing the tissues in broth, brain-heart infusion and a special medium for anaerobes. No growth was obtained in 13 cases; in one staphylococcus albus was isolated. The absence of bacteria is ascribed to the antibiotic therapy given before enucleation. The continued presence of an inflammatory reaction is probably due to allergic phenomena after a sensitizing infection.

John J. Stern.

von Sallmann, L., Jones, I. S., Wiggins, R. L., and Locke, B. D. **The influence of cortisone on wound healing, macrophagocytosis and capillary permeability.** Wien. klin. Wchnschr. **63**:684-686, 1951.

The re-epithelization of the cornea of rats was slightly inhibited by cortisone applied after superficial trauma. Local application of cortisone also slows the healing of trephine defects in the sclera of monkeys by inhibition of the formation of granulation tissue and its transformation into dense scar tissue. The fistulizing activity of these scars is only briefly prolonged by cortisone. Parenteral injection of cortisone hastens the absorption of autogenous red blood corpuscles injected into the vitreous of rats. Increased macrophagocytosis is probably responsible for this phenomenon. The permeability of the blood-aqueous barrier for fluoresceine seemed to be slightly less in cortisone-treated rabbits than in normal animals. (3 figures, references)

Max Hirschfelder.

Segal, Pawel. **Experimental diagnosis of virus diseases of the eye.** Klinika Oczna **20**:304-318, 1950.

Segal described his experimental inoculations of animals and chicken embryos with material taken from 11 patients with various diseases of the anterior segment of the eye. Inoculations were made into the cornea of rabbits, the brain of white mice and the chorion allantois of chicken embryos. It was possible to discover virus infections even when a bacterial infection was superimposed. Virus infection superimposed on an old tuberulous process may appear merely as the exacerbation of the old condition. Corneal scarification was the most useful diagnostically because of ease of observation. At the same time the virulence of the strain was increased and it was easier to obtain positive results with the other methods. (8 figures, 1 table)

Sylvan Brandon.

3

VEGETATIVE PHYSIOLOGY, BIOCHEMISTRY, PHARMACOLOGY, TOXICOLOGY

Asboe-Hansen, G., and Iversen, K. **Influence of thyrotropic hormone on connective tissue: pathogenetic significance of mucopolysaccharides in experimental exophthalmos.** Acta Endocrinol. **8**:90-96, 1951.

The subcutaneous injection of large doses of thryotrophin into guineapigs results in a general fat mobilization and an accumulation of fat in the liver, muscles, and kidneys within 24 hours.

By means of a special histochemical technique the authors demonstrated accumulations of mast cells and hyaluronidase-sensitive mucopolysaccharide in the connective tissue at various sites, most

marked in the retrobulbar tissue, of guineapigs treated with thyrotrophin. Since it is known that mucopolysaccharides, particularly hyaluronic acid, posses a marked capacity to bind water, an increased content of these substances may explain the protrusion of the eyes.

Theodore M. Shapira.

Cima, V. **Experimental thyrotropic exophthalmos and vitamin E.** Boll. d'ocul. **30**:342-353, June, 1951.

Two groups of eight guinea pigs each were given thyrotropic hormone over a period of 20 days until their weights decreased by 20 percent. Later the amount was calculated in international guinea pig units. Every other day, the animals of one group only received additional alpha-tocopherol, 30 mg. per injection, for one month. There was no appreciable difference in exophthalmos between the two groups during and after the treatment period. Exophthalmos was measured by ascertaining the distance between the vertex of the right and left cornea and differences up to 10 percent were not considered to be significant. Further experiments included thyroidectomy, administration of thiouracil, with and without additional administration of vitamin E. No significant difference between the controls and the animals receiving vitamin E could be ascertained; the exophthalmos was most marked in thyroidectomized animals.

K. W. Ascher.

Kapuscinski, W. J., and Zebrowski, J. **External hypervitaminosis A in white rats.** Klinika Oczna **20**:328-331, 1950.

This is a continuation of work published in 1938. Vitamin A (100,000 units per cc.) was instilled into the eyes of 32 white rats, and it was found that the hypervitaminosis caused a change of color and falling out of the hair, slight baldness of the lids and slight damage to the corneal epithelium. Addition of vitamin D to the vitamin A prevented these changes.

Sylvan Brandon.

Marconcini, E. **The action of nicotinamide on the optic nerve of animals (rabbits) poisoned with quinine (monochlorhydrate).** Arch. di ottal. **55**:355-362, May-June, 1951.

In a previous paper the author had reported a direct metabolic effect of nicotinamide on the optic nerve of animals poisoned with quinine. In order to prove that the improvement seen histologically was not due to natural recovery he repeated the experiment and omitted treatment with nicotinamide. Three rabbits were given a total of 20 to 24 centigrams of quinine monochlorhydrate intramuscularly over a period of 10 to 12 days, after which one eye was enucleated. After an equal period of time the other eye was also enucleated. Histological studies demonstrated a degeneration of the myelin fibers of the optic nerve in both eyes. Therefore the improvement seen in the previous experiment must have been due to treatment and not to spontaneous recovery.

John J. Stern.

Remky, H. **Remarks to Weiglin and Freusberg's proposals concerning standardization of dynamometry.** Ophthalmologica **122**:18-30, July, 1951.

Weiglin and Freuberg's paper was reviewed in the Am. J. Ophth. 34:473, 1951. Remky's remarks concern themselves primarily with the effects of postural changes upon retinal circulation. Specifically, the paper deals with the study, by means of dynamometry, of the regulatory mechanisms that become operative during postural changes.

Peter C. Kronfeld.

Schubert, G. **The control of the functions of extraocular muscles.** Wien. klin. Wchnschr. **63**:632-34, 1951.

With the head fixed, binocular movements enlarge the optically visible space. These conjugated muscle movements are tetanic in nature. Their central nervous regulation originates in the frontal cortical field, which, from an embryonic standpoint, belongs to the pyramidal system. Optic impressions from outer space lead, after a relay in the occipital cortex, to a tonic reaction of the extraocular muscles. In monocular vision these tonic impulses govern the fixation reflex of the fovea and are responsible for the fusion reflex of binocular vision. The mechanism is the same in man as in the lower animals.

Max Hirschfelder.

Sobański, J., Miratyńska-Ernestowa, E., and Janiszewski, S. **The influence of direct pressure on the eyeball on the intraocular tension.** Klinika Oczna **20**:231-236, 1950.

The curves used for transposing the findings of the Bailliart's dynomometer into the mg. Hg, when measuring blood pressure of the central retinal vessels, are only applicable for people with normal blood pressure and are not correct for hypertensives. The manometric pressure inside the eye was checked against the tonometers of Schiøtz and Fick; then the dynamometer of Sobanski was used and both pressures were plotted. The curve obtained presents a straight line with a lower angle than the curve of Muller or Bailliart. The authors believe they have shown that the pressures in the central retinal vessels are really lower than they have been considered until now. (4 tables)

Sylvan Brandon.

de Vincentiis, M. **Histochemical contribution to the study of the phosphatases in the ocular tissues.** Arch. di ottal. **55**: 303-328, May-June, 1951.

Alkaline phosphomonoesterase is concentrated in the choroid, the ciliary body and the posterior layer of the iris. An acid phosphatase has not been found anywhere in the ocular tissues. The original paper should be consulted for methods and details.

John J. Stern.

4

PHYSIOLOGIC OPTICS, REFRACTION, COLOR VISION

Amsler, Marc. **Quantitative and qualitative vision.** Tr. Ophth. Soc. U. Kingdom **69**:397-410, 1949.

When the author uses his charts to outline an abnormality of the central fields he asks the following questions. Do you see the white spot in the center of the square? Do you see the corners and sides of the large square? Is the canvas of the larger square intact, or does it show interruptions, holes or blemishes? If so, where? Are all vertical and horizontal lines straight and parallel? Are the small squares equal to each other? Do you see movement or vibration of certain lines? Do you see luminous phenomena, colors or tints? How many intact squares are there between the fixation point and the spot or deformation that you see? In this way the function of the macular area can be determined and metamorphopsia in all its varieties in the beginning of macular disease can be outlined. For treatment of early lesions he advised the use of different dilators together with calcium therapy and ascorbic acid. Beulah Cushman.

Cabarrouy. **Precorneal lenses.** Arch. d'opht. **11**:470-473, 1951.

The author lists the many disadvantages of the conventional contact glasses and advocates the use of corneal lenses which contact the cornea only, require no fluid, are almost invisible, and do not interfere with ocular movements. In prescribing the lenses only three measurements are required: the ophthalmometer readings, the vertical and horizontal diameters of the cornea, and the refraction. High degrees of ametropia and certain cases of keratoconus have been success-

fully fitted. Toleration of the lens by the cornea is generally good, but must be developed. The lenses are maintained in position by capillary attraction. A few cases of high myopia have been troublesome because of the necessarily thick edges of the lenses. Phillips Thygeson.

Hurvich, L. M., and Jameson, D. **The binocular fusion of yellow in relation to color theories.** Science **114**:199-202, Aug. 24, 1951.

Hecht's demonstration of the binocular fusion of yellow from monocular red and green stimuli has been frequently accepted as a crucial experiment for judging the validity of the two major classes of color theory, the three-component and the four-component theories. However, the experiment is crucial only if it is assumed that monochromatic red stimulus evokes nothing but a red sensation and the green a green sensation. Actually pure physical stimuli do not necessarily evoke pure hue sensations. The psychological relation between hue and wave length is a complex one and the authors present an analysis that clarifies the meaning of these experiments. The real problem is to provide an understanding of the nature of the process whereby the central "cancellation" occurs when a binocular mixture of pure red and green stimuli results in a neutral sensation and a mixture of yellow-red and yellow-green results in yellow. Neither the red nor the green of the monocular stimulus appears in the resulting sensation.

F. H. Haessler.

Pendse, G. S., Dan De Kar, V. M., and Bhave, L. S. **Preliminary note on study of the refractive state with special reference to myopia.** A.M.A. Arch. Ophth. **45**: 168, Feb., 1951.

Five hundred and ninety unselected school children in Deccan (India) were refracted and the heights, weights and blood pictures were recorded. The frequency curve plotted for this series shows the mean refraction to be +1.0D, the mole +1.25D and the standard deviation +1.0D. The curve for variation of refraction according to age indicates a definite downward trend. The average refraction at the age of 7 years was +1.5D, whereas at the age of 13 it was +0.5D. There is evidence that subjects with subnormal heights have higher (more hyperopic) refractions, while those who are taller for their ages have lower refractions. The state of refraction seems to be determined mainly by factors concerned with body growth. John C. Long.

Pike, J. **Some problems in ophthalmic dispensing.** Tr. Ophth. Soc. U. Kingdom **69**:265-282, 1949.

To avoid risk of misinterpretations by the optician, the distance of the trial lens from the cornea should always be given, especially with the stronger lenses. Tables for the variation at the different distances are given. Decentration is limited by the approach of maximum lens size to standard lens blank size. Distortion is reduced in the corrected lenses and the tilting of lenses with respect to the visual axis may vary from 5^{Δ} to 15^{Δ} and should be calculated. Lenticular lenses are advised for cataract glasses.

Beulah Cushman.

Popkowski, Jan. **Range of fusion tested with stereoscopic moving pictures.** Klinika Oczna **20**:86-96, 1950.

The author presents a short summary of the literature on fusion and problems connected with it. He describes methods of examination and presents the results of examination of 50 individuals with stereoscopic pictures, stationary and cinematographic. The range of fusion was greater by 44 percent with cinematographic stereoscopic pictures than with immobile ones. Sylvan Brandon.

Santoni, A. **The shape of the optic nerve head in the astigmatic eye.** Boll. d'ocul. 30:329-341, June, 1951.

With the patient's head fixed, and the ophthalmoscopic lens of 13 diopters consistently held at the same distance and exactly parallel to the patient's face, Santoni compared the longest and shortest diameters of the papillae, using a millimeter graticule visible on the plane surface of his planoconvex ophthalmoscopic lens. A table shows the relationship between the vertical and horizontal diameters of the discs of 50 emmetropic eyes; in 38 of them the quotient was between 0.9 and 1.11; 2 had an 0.8 and 2 a 1.33 quotient. Twenty eyes with myopic astigmatism with the rule, 8 with myopic astigmatism against the rule, 20 with hypermetropic astigmatism with the rule and 5 with hypermetropic astigmatism against the rule, 8 eyes with mixed astigmatism with the rule and 5 eyes with mixed astigmatism against the rule were measured and much larger deviations from the quotient 1 were found; the greatest was 1.5 in inverse hypermetropic astigmatism and inverse mixed astigmatism, and 0.6 in mixed astigmatism with the rule. Thus, the disc appears to be flattened according to the meridian of higher refraction; the main meridians of the ellipse of the disc sometimes coincide with the main axes of the corneal astigmatism. (7 tables)

K. W. Ascher.

Scullica, Francesco. **The limits of stereoscopic fusion.** Ann. di ottal. e clin. ocul. 77:303-322, Aug., 1951.

Scullica measured the limits of stereoscopic fusion in ten subjects by experiments with Holmes's stereoscope, with Maggiore's mirror stereoscope, and without any stereoscope (by dissociation of accommodation and convergence). For stereograms he employed the familiar pair of two eccentric circles, and determined the limits of fusion by noting how far the inner circle of each pair could be displaced in various directions while stereopsis was maintained. He concludes that there are no absolute limits within which stereoscopic fusion is possible. The limits are imposed by optical and anatomicophysiologic factors (visual acuity, region of the retina concerned, interpupillary distance) which are more or less constant for all subjects at all times, and by psychologic factors (as nystagmiform movements of the eyes, suppression, attention, stereoscopic training) which vary from subject to subject and in the same subject under different conditions. No sharp boundary can be drawn between the optical and the psychologic limits. If the binocular parallactic angle is equal to or is less than the minimum visual angle of one minute, the optical factors are chiefly operative, but if the minimal visual angle is exceeded, fusion generally does not occur without intervention of psychologic factors. The triad playing the principal role in determining the limits of stereoscopic fusion are the unvarying stereoscopic parallax and the variable attention and ocular movements. (References)

Harry K. Messenger.

Ten Doesschate, G. **The subjective visual space under the influence of perspective.** Ophthalmologica 122:46-50, July, 1951.

The object of this study was to analyze and define the subjective visual space which a visually normal, binocular observer unconsciously constructs around himself while viewing a drawing or painting made in accordance with the rules of perspective. The tentative conclusion is that even under these conditions our subjective visual space remains strictly subjective and distinctly different from a space based on Euclidean geometry.

Peter C. Kronfeld.

5

DIAGNOSIS AND THERAPY

Armstrong, S. H., Jr., and Irons, E. N. **Physiological considerations of ACTH and cortisone therapy with reference to ophthalmology.** A.M.A. Arch. Ophth. **45**: 251-257, March, 1951.

ACTH and cortisone are described chemically and biologically and their physiological properties are discussed in relation to their use in ophthalmology. Cortisone restores normal glucose tolerance in Addison's disease and can prevent hypoglycemia. In normal subjects prolonged administration of large doses leads to a diabetic state, which usually disappears when therapy is stopped. When ACTH is given to a patient, the adrenal gland secretes a series of hormones. Thus, there is a salt-retaining effect, a potassium loss effect, a cerebral effect, and an effect on glucose tolerance, all of which become important in long term therapy. The problems of clinical balance are not as important in ophthalmology as in clinical medicine because: 1. the eye can be influenced by local therapy, and effective high local concentrations of cortisone have been achieved to give remarkable therapeutic results without sufficient general absorption to produce untoward metabolic effects, and 2. the diseases in which therapy has been most effective have been sufficiently cyclical or self limited, so that the prolonged courses of medication often needed in internal medicine can be avoided.

The effects of cortisone, briefly reviewed, are as follows:

1. Cellular exudation is reduced. 2. The permeability of the capillaries to proteins in regions of inflammation is reduced. 3. The development of an intense fibrotic reaction in the course of healing is lessened. Although these results are beneficial, the weakening of fibrous repair can be deleterious to the healing of surgical wounds in patients under intensive ACTH or cortisone therapy. Local cortisone therapy is suitable for the eye and these deleterious effects can, in general, be avoided.

E. J. Swets.

Azzolini, U., and Faldi, S. **Oidiomycine 101 in ocular therapy.** Arch. di ottal. **55**: 285-292, May-June, 1951.

Oidiomycine 101 is an extract from oidium lactis which has certain constant biochemical characteristics. In its therapeutic effect it acts as a catalyst and biological stimulant. It was given subconjunctivally and parenterally and was effective in certain cases of keratitis and particularly in four cases of anterior uveitis.

John J. Stern.

Belz. **The ultrasounds in ophthalmology.** Ann. d'ocul. **184**:673-678, Aug., 1951.

Ultrasonic therapy is based on the premise that the mechanical vibrations, if properly applied to tissues, will hasten the absorption of some inflammatory exudates. The generator resembles that used in developing high frequency currents. The physico-chemical energy thus developed is transmitted to the tissues through a quartz rod with or without the further protection of rubber or vaseline. The current frequency should be between 800-1,000 Kc/sec. and the applications last from one to five minutes. Two or three treatments weekly are given, usually for several weeks. Several cases of uveitis, deep keratitis and vitreous hemorrhage treated with this therapy are described and seem to have progressed favorably.

Chas. A. Bahn.

Best, F. **The treatment of acute lime burns.** Ophthalmologica **122**:58-63, July, 1951.

On the basis of theoretical considerations and practical experiences in a few cases, the author recommends that the

first aid in lime burns include repeated instillation of 1-percent hydrochloric acid. This treatment should be administered by ophthalmologist, general practitioner, industrial nurse or fellow worker.

Peter C. Kronfeld.

Biegel, A. C. **Effect of cortisone on horse serum uveitis in rabbits.** A.M.A. Arch. Ophth. **45**:258-273, March, 1951.

The uveitis produced by the intravitreal injection of horse serum in previously sensitized rabbits was treated by the systemic administration of cortisone. Daily administration of cortisone intramuscularly had a suppressing influence on most of the features of horse serum uveitis in rabbits. Its beneficial effect was probably the result of inhibition of the inflammatory reaction itself rather than a reduction in the systemic hypersensitivity to horse serum. (7 figures, 1 table)

E. J. Swets.

Brückner, R. **Experiences with cortisone applied locally.** Ophthalmologica **121**:275-286, May, 1951.

From the observation of 29 cases of various forms of ocular inflammations treated topically with 1-percent cortisone suspension or ointment, the author draws the conclusion that cortisone is indicated in the acute, "toxic-allergic" inflammations of the anterior segment, especially if they entail a disturbance of the water-exchange between the cornea and adjoining fluids. Cortisone is contraindicated in keratitides due to bacterial invasion, in recent corneal scars and in tuberculous diseases of the eye. Peter C. Kronfeld.

Coppez, Léon. **Improvements in surface coagulation by means of heatmeter electrode (detachment of the retina and cyclodiathermy).** Tr. Ophth. Soc. U. Kingdom **69**:535-547, 1949.

The instrument described is 2.5 mm. in diameter and in 20 seconds raises the temperature to 80°C. This will produce the same scar under all conditions. By using this control Coppez has been able to avoid numerous accidents associated with diathermy and for 17 years has found surface coagulation with the heatmeter electrode to be most satisfactory.

Beulah Cushman.

Douvas, N. G., Featherstone, R. M., and Braley, A. E. **Role of terramycin in ophthalmology.** A.M.A. Arch. Ophth. **46**: 57-68, July, 1951.

Ophthalmic solutions of terramycin hydrochloride were more stable than aureomycin in the same solution, and remained stable for five days when stored at room temperature. Terramycin compared favorably with the other antibiotic and chemotherapeutic agents studied when used experimentally in rabbit aqueous humor but failed to protect mice inoculated with the virus of herpes simplex. Clinically, terramycin was well tolerated and effective in the treatment of conjunctivitis and corneal ulcerations of bacterial origin; it was not effective in the treatment of acute follicular conjunctivitis, type Beal, or in dendritic keratitis. One-tenth-percent terramycin hydrochloride ophthalmic ointment was effective when used before operation for the prophylactic removal of bacteria from the conjunctival sac. (6 tables) Edward J. Swets.

Fitzgerald, J. R., Bellows, J. G., Donegan, J. M., Gamble, R. C., Krause, A. C., Mann, W A., Pearlman, M. D., and Zekman, T. N. **Early clinical results of ACTH and cortisone treatment of ocular diseases.** A.M.A. Arch. Ophth. **45**:320-333, March, 1951.

The largest eye clinics in the Chicago metropolitan area agreed to pool their initial experiences with ACTH and cortisone and present the results primarily from the point of view of practical clinical ophthalmology. The daily dose of ACTH

ranged from 40 to 75 mg. and was given for from seven to ten days. Cortisone was most frequently given in single daily injections of 100 mg. for an average of 10 days. In most instances 25 mg. was used locally in isotonic sodium chloride solution diluted 1 to 4, and one drop was instilled every hour or two.

Clinical improvement was judged by subsidence of subjective manifestations of the disease, clearing of the media, resolution of ocular hyperemia and by-products of the inflammation, and unequivocal visual improvement. The greatest therapeutic efficacy of ACTH and cortisone exists in treatment of the acute exudative inflammatory lesions of the uveal tract, in which they produce a rapid, and frequently dramatic, amelioration of the signs and symptoms of the disease, and recovery which is well sustained. Local administration of cortisone in the few cases reported compares favorably with the systemic use of ACTH or cortisone in inflammations of the anterior segment. Chronic inflammations of the uveal tract respond less well than do the acute types. In such external diseases as episcleritis, sclerokeratitis, phlyctenulosis, dendritic ulcer and rosacea keratitis, the results of local cortisone therapy have been excellent; in other external conditions such as allergic disorders and marginal ulcer, further clinical experience is necessary. In a variety of miscellaneous ocular conditions and diseases of the retina and optic nerve, no therapeutic aid was evident. (10 tables, 6 figures) E. J. Swets.

Gandolfi, C., and Morpurgo, F. **Thiosemicarbazone (TB 1-698) in the treatment of ocular tuberculosis.** Arch. di ottal. 55:293-302, May-June, 1951.

Eight cases of tuberculous iridocyclitis, three of keratitis and two of retinal periphlebitis were improved or cured with thio-semicarbazone, a new compound constructed by Domagk. Two cases of nodular tuberculous iritis, one of iridocyclitis and two of periphlebitis did not respond.

John J. Stern.

Haselmann, G., and Schmidt, E. **Comparison of light sources for red-free ophthalmoscopy.** Ophthalmologica 121:334-344, June, 1951.

The spectral composition of the light emitted by Vogt's original source, that is, the carbon arc with a fluid or glass filter, is compared with the output of modern naturally red-poor mercury arc lamps. The main difference between these two sources is the high yellow content of the light emitted by the mercury arc.

Peter C. Kronfeld.

Hruby, K. **The development and present status of biomicroscopy of the eye.** Wien. klin. Wchnschr. 63:669-672, 1951.

The original slitlamp by Gullstrand opened the way for biomicroscopy of the anterior segment of the living eye. Examination of the posterior segment was first attempted by Koeppe in 1918 by means of an additional silver mirror which deflected the slitlamp beam and a flat contact glass which eliminated the corneal refraction and made observation of the fundus possible. Goldmann substituted a "reduction prism" for the mirror and described a more suitable contact glass. The strong concave lens by Hruby makes the use of the contact glass unnecessary. The most modern slitlamps (Haag-Streit, Zeiss-Opton) have an observation and an illumination system which are mechanically and optically interlocked. They can be handled with one hand, make possible the reduction of the angle between the axis of illumination and the axis of observation without additional apparatus, and are equipped with the Hruby lens and fixation light for observation of the posterior segment. The article closes with the enumeration of the various conditions which can be diagnosed by biomicroscopy

of the posterior segment in the vitreous as well as in the retina. Gonioscopy and arc light biomicroscopy are shortly discussed. (2 figures) Max Hirschfelder.

Lepri, G., and Montanari, L. **The effect of the corticotropic hormone (ACTH), desoxicorticosterone and adrenal cortex extract on the hyaluronidase content of the aqueous.** Arch. di Ottal. 55:329-340, May-June, 1951.

Administration of ACTH produces a diminution of the hyaluronidase concentration in the aqueous after one injection, which was more marked after prolonged use. The total extract of the adrenal cortex showed the same effect to a lesser degree; desoxicorticosterone had no effect at all. John J. Stern.

Mosher, H. A. **Local use of cortisone in ophthalmic diseases.** A.M.A. Arch. Ophth. 45:317-319, March, 1951.

The observations on cortisone used locally in 53 patients at the Massachusetts Eye and Ear Infirmary are recorded. Local instillation was preferred because: 1. there is little danger of systemic reaction; 2. the cost to the patient is less; 3. hospitalization is not necessary; 4. local treatment could probably be continued almost indefinitely; 5. a high local concentration is possible. In this series cortisone was more effective when administered as a collyrium than when injected subconjunctivally. It was more effective in diseases of the anterior segment than in those of the posterior segment of the globe. The collyrium was definitely effective in treatment of sclerokeratitis, superficial punctate keratitis, bullous keratitis and relapsing keratitis. Cortisone was not effective in the treatment of Eales's disease or rodent ulcer of the cornea or in one fourth of the patients with uveitis. One patient with severe chorioretinitis showed no benefit after intensive local use.

E. J. Swets.

Olson, J. A., Steffensen, E. H., Smith, R. W., Margulis, R. R., and Whitney, E. L. **Use of adrenocorticotropic hormone and cortisone in ocular disease.** A.M.A. Arch. Ophth. 45:274-300, March, 1951.

The authors believed that the benefit derived from nonspecific foreign protein therapy could be attributed in part to the release of endogenous adrenocorticotropic hormone and its acceleration of mechanisms normally evoked in stress states. The results of its use in the treatment of 51 patients with a wide variety of ocular lesions are reported.

Excellent results were obtained in the group with acute inflammatory lesions, and rapid and complete healing resulted in most cases. Here, both ACTH and cortisone gave comparable results and appeared superior to the currently employed methods of therapy. In certain cases of inflammation of the anterior segment of the eye, relapses were successfully treated with topical instillations of cortisone. Chronic inflammatory diseases showed a definite, but transitory, response under the currently used brief intensive method of therapy. In this group the value both of interrupted and of sustained, less intensive therapy is now under investigation.

No improvement was seen in degenerative disease, primary glaucoma and optic nerve atrophy. In retinitis pigmentosa a limited response occurred in two patients. In one case of vernal conjunctivitis there was a striking but temporary improvement. E. J. Swets.

Pulvertaft, R. J. V., Duthie, O. M., Goldsmith, A. J. B., Philps, A. S., Mann, I., Purvis, V., Foster, J. **Discussion of principles and practice of asepsis in ophthalmic operations.** Tr. Ophth. Soc. U. Kingdom 69:355-395, 1949.

Pulvertaft, R. J. V. pp. 355-364.

The risk of infection in ophthalmic work is low except in cataract operations. The hypodermic syringe is the most dan-

gerous implement. The following precautions are advised. The air should be sterilized by use of ultraviolet light or with aerosols. Dust should be controlled by air conditioning and the use of caps and masks and there should be no talking. Chemical sterilization by immersion of instruments in 1 to 3,000 zephiran has been found satisfactory. Each hospital bacteriologist should check the surgical technique at least once a month.

Duthie, O. M. pp. 365-374.

Sterilization of instruments can be completely aseptic, but when prepared drops and sutures are used asepsis should be frequently checked. The lacrimal sac should be carefully examined and extirpated if there is any question that it may harbor infection. Gloves should be worn. The incidence of infection in Duthie's work was approximately 0.6 percent over a 10 year period. The highest incidence was in cataract surgery with 14 patients out of 996.

Goldsmith, A. J. B. pp. 375-380. Goldsmith discusses the factors of resistance to infection inherent in the patient.

Philps, A. S. pp. 381-383.

Hot air sterilization of instruments in racks is considered the best form of sterilization. The disadvantages are that many sets of instruments are required in a busy clinic but instruments can be kept sterile and ready for use in special containers. The surgeon is advised to avoid letting sutures drag over the skin.

Mann, I. p. 384.

A critical review of the data in 60 consecutive operations for cataract confirms the author's belief that clinical judgment is certainly as safe as and possibly a little safer than culturing.

Purvis, V. pp. 385-387.

The main barriers to infection are a small wound, which is not touched, and instruments which are dry. Sutures should not be touched, nor catgut used and the eye should be isolated from the surrounding skin. Breakdowns in aseptic technique occur in irrigation of the anterior chamber, and during prolonged manipulations which involve too many instruments.

During a three-year period the author did not wear gloves, washed his hands for two minutes only, then rinsed them in cetavlon, did not wear a mask or hat, and did not use catgut. A culture was made in every case and if the results were suspicious clinically the eye was irrigated with 1 to 1,000 silver nitrate solution. The anterior chamber was never irrigated. During this time there were three cases of sepsis, two of them in mental patients who pulled off their bandages and tried to remove the sutures, the third in a patient with squint in a general hospital where care had been inadequate.

Foster, J. pp. 389-395.

The author reports seven cases of infection in 673 cataract operations. Cultures had been made in all and a high percentage showed "dirty cultures." One patient had an undetected mucocele and one a facial impetigo. Beulah Cushman.

6

OCULAR MOTILITY

Burian, H. M. **Strabismus annual review.** A.M.A. Arch. Ophth. **46**:73-93, July, 1951.

In this paper the 1950 literature on strabismus is reviewed in detail and the etiology, symptomatology, non surgical and surgical treatment are discussed.

Edward J. Swets.

Moschik de Reya, N., and Eichhorn, O. **Registering bio-electric currents in ocular movements.** Wien. klin. Wchnschr. **63**: 676-677, 1951.

Action currents of ocular muscles could be demonstrated through the amplifying system of an instrument used for encephalography, which enabled graphic

registration of the electric potential differences in the action currents of ocular muscles. The method can be used for an analysis of voluntary eye movements, for objective registration of paresis of the extraocular muscles and for the differentiation of various kinds of nystagmus. (3 graphs) Max Hirschfelder.

7

CONJUNCTIVA, CORNEA, SCLERA

Dean, A. M. **Physiology of the conjunctival sac.** J. Iowa St. M. Soc. **41**:459-460, Nov., 1951.

Burning of the eyes is a common complaint and often greatly magnified in the patient's mind. Anything which tends to keep the eyes open under strain, also smoke, dust, fumes, allergies, all these tend to hasten the drying reflex and make the patient more uncomfortable. Careful explanation will reassure the patient and perhaps the ingestion of more water may be of help. Bennett W. Muir.

Leahey, B. D. **Bullous keratitis from vitreous contact.** A.M.A. Arch. Ophth. **46**:22-32, July, 1951.

In uncomplicated intracapsular cataract extraction, herniation of the vitreous into the anterior chamber frequently occurs. When the vitreous remains in contact with the posterior surface of the cornea, permanent intractable bullous keratitis may arise.

The location, duration, and density of the contact and the condition of the corneal endothelium at the area of contact bring this about. An effective method of treatment consists of 1. release of a generous amount of vitreous by posterior sclerotomy; 2. freeing of vitreous adherent to the cornea by a spatula introduced as for a cyclodialysis, and 3. injection of a large air bubble into the anterior chamber. This procedure, combined with wide cyclodialysis, also gives excellent results in aphakic eyes without vitreous contact, in which an extensive anterior synechia and secondary glaucoma exist.

Edward J. Swets.

Lindner, K. **Keratoplasty.** Wien. klin. Wchnschr. **63**:627-628, 1951.

This operation was first performed by Reisinger in Vienna in 1818, and v. Hippel in 1887 and Fuchs in 1894 reported poor results as their grafts did not remain clear. Zirm had a successful case in 1905 and thereafter techniques improved and results were more satisfactory. Lindner quotes the statistic of Löhlein who obtained healing grafts in 92 percent of his 286 plastic operations. Cases of keratoconus were most successful, with improvement in 83 percent. In interstitial keratitis there was improvement in 53 percent. Considering all operated eyes, keratoplasty gives promise of definite visual improvement in 33 percent of half the cases and in 50 percent of a quarter of the cases.

Max Hirschfelder.

Maddin, S., and Danto, J. L. **Results of topical application of cortisone in congenital syphilitic interstitial keratitis; preliminary report of two cases.** A.M.A. Arch. Derm. and Syph. **64**:437-440, Oct., 1951.

The treatment of congenital interstitial keratitis has been very unsatisfactory. Two cases are reported in which this condition was treated by the topical application of cortisone. The first patient was a 15-year-old Puerto Rican girl, the second a 13-year-old Negro girl. In the first patient treatment was started within two months of the onset of ocular symptoms, in the second not until after nine months. The results in the first case were more striking than in the second. Penicillin, with or without artificial fever therapy, remains the method of choice in the treatment of congenital syphilis, but cortisone may be a useful addition in interstitial keratitis. Bennett W. Muir.

Nagy, F. **The treatment of burns of the eye.** Ophthalmologica **121**:345-352, June, 1951.

In 30 cases of corneo-conjunctival burns due to various caustic agents, the local application of the vasodilator priscol (instillations of 10-percent aqueous solution 3 to 5 times daily were given) gave very satisfactory results. In cases of extensive necrosis of the conjunctiva the author practices and recommends Denig's operation, which consists of surgical removal of the necrotic tissue and replacement with mucous membrane from the mouth.

Peter C. Kronfeld.

Radnót, M. **A virus disease of the eye.** Ophthalmologica **121**:287-289, May, 1951.

Four cases of acute conjunctivitis with marked preauricular lymphadenitis due to infection with the Newcastle virus of fowls (cfr. Am. J. of Ophth. 30:243, 1947) are described. Peter C. Kronfeld.

Valière-Vialeix. **Pterygium operations by means of free graft of the conjunctival mucosa or pedicle flap implants. Reflections on the pathology of pterygium.** Ann. d'ocul. **184**:811-825, Sept., 1951.

Pterygium operations are based on three patterns of procedure, namely physical or chemical destruction, change of direction by folding or other means, and resection. These may be used without or with grafts or plasties. In plastic procedures the denuded conjunctiva is covered with a plasty or flap usually taken from the conjunctiva or the mucous membrane of the lip. The technique recommended by the author consists of separation of the pterygium from the cornea with a Graefe knife; scarification of the involved cornea and episclera; resection of the pterygium; insertion of a plasty from the upper temporal conjunctiva, or a pedicled flap from the upper nasal conjunctiva to cover the denuded area; and fixation with silk sutures.

Chas. A. Bahn.

8

UVEA, SYMPATHETIC DISEASE, AQUEOUS

Berliner, M. L., and Breinin, G. M. **Angioma of the choroid.** A.M.A. Arch. Ophth. **46**:39-48, July, 1951.

Angioma of the choroid is the basic ocular lesion of the Sturge-Weber syndrome (encephalotrigeminal angiomatosis). It also occurs as an isolated tumor, which is visible ophthalmoscopically but without cutaneous vascular nevi. These tumors must be distinguished from malignant melanomas. Angiography may be useful in outlining vascular tumors of the eye. The authors describe a case of Sturge-Weber syndrome with buphthalmos and one of isolated choroidal angioma which appeared as a tumor in the fundus, and give the pathologic findings on the enucleated eyes. (8 figures)

Edward J. Swets.

Czukrász, Ida. **Atypical colobomas of the iris.** Ophthalmologica **122**:53-57, July, 1951.

Four cases of atypical congenital colobomas of the iris are reported and illustrated. They are interpreted as the result of the same mechanism, that is, persistence of iridohyaloid vessels which produce dents in the margin of the optic cap.

Peter C. Kronfeld.

François, Jules. **Rubeosis iridis.** Ophthalmologica **121**:313-333, June, 1951.

Three cases of typical rubeosis iridis in patients with diabetes are reported in detail. The disease is characterized by a pathological vascular proliferation which makes its visible start either on the pupillary portion of the iris or in the chamber angle. The glaucoma which is a typical feature of the disease, is attributed to vascular changes in the uvea. The clinical picture and the pathogenesis of rubeosis iridis are entirely different from those of glaucoma secondary to central retinal

vein destruction. Treatment is unsuccessful and the visual prognosis is bad.

Peter C. Kronfeld.

von Sallmann, L., Locke, J. C., and Locke, B. D. **Cytological and bacteriological studies of the aqueous humor in uveitis.** A.M.A. Arch. Ophth. **46**:4-13, July, 1951.

Swiss investigators, Amsler, Verrey, and others reported in 1946 that 20 percent of the smears from centrifuged samples of the aqueous from 589 patients with uveitis showed phagocytosed bacteria, and growths had been obtained on culture in from 3 to 6 percent of 302 samples. Combinations of cytological and bacteriological tests were used in the present work which, in all essential respects followed the method of Amsler and Verrey. The most important observation was the absence of free or phagocytosed bacteria in the smears of centrifuged aqueous in all but one patient. The 4-percent incident of growth-yielding cultures in this series was comparable to the Swiss investigation. Cytological and bacteriological examinations of the aqueous humor in endogenous uveitis without septicemia may eventually prove its etiology.

Edward J. Swets.

Weekers, R., and Prijot, E. **Leukemic uveitis.** Ophthalmologica **122**:1-9, July, 1951.

A typical case of bilateral uveitis due to lymphatic leukemia is reported and illustrated in detail. An anatomical as well as functional cure was achieved by means of contact X-ray therapy.

Peter C. Kronfeld.

9

GLAUCOMA AND OCULAR TENSION

Weekers, R., and Prijot, E. **The relationship between the ocular tension and the caliber of the aqueous veins.** Ophthalmologica **121**:264-274, May, 1951.

The effect of topically applied adrenalin upon the caliber of the aqueous veins and, consequently, upon the ocular tension was studied in rabbits. The application of the drug, by means of a small cotton applicator, to a single laminated vessel caused marked localized constriction and the appearance of collaterals not visible before. The ocular tension remained unchanged. If adrenalin was instilled into the cul-de-sac and therby made to act on a large number of vessels, very general constriction of the epibulbar vessels ensued, associated with a rise in ocular tension from 15 to 25 mm. Hg Schiøtz. The two phenomena ran a very similar course but were of different magnitude. The rise of tension was of fairly constant height, while the visible degree and extent of vasoconstriction varied widely. This discrepancy suggests a pressure-regulating mechanism in the form of reserve outlets deep in the sclera and therefore uninfluenced by topically applied adrenalin. The possibility of a regulatory inhibition of the aqueous formation is also considered.

Peter C. Kronfeld.

10

CRYSTALLINE LENS

Rotter, H. **Improvement of vision in aphacic eyes by the use of Mintacol and without lenses.** Wien. klin. Wchnschr. **63**: 683-684, 1951.

The use of the miotic Mintacol, a parasympathetic-stimulating drug, brought about contraction of the round pupil in eyes recently operated upon for cataract to an average of 1.5 mm. This constriction led to improvement of the aphacic uncorrected vision to an average of about 1/20 visual acuity. The drug may be used during the initial period, before glasses can be given.

Max Hirschfelder.

Ridley, Harold. **Intra-ocular acrylic lenses after cataract extraction.** Lancet 1: 118-121, Jan. 19, 1952.

All the disadvantages of aphakia except the lack of aphakia can be overcome by inserting an inert artificial lenticulus into the eye at the time of extraction of a cataract. A plastic polymethyl methacrylate named perspex or plexiglass was chosen because it is light and tolerated by other tissues. It can be sterilized by immersing it for half an hour in 1-percent cetrimide solution. The first intra-ocular lenticuli were designed to approximate the dimensions and curvature of the unaccommodated human lens. The diameter was made 1 mm. less than that of the lens to make insertion easier and to avoid pressure on the ciliary body and the region of the filtration angle. These lenses proved to be too strong optically. Now the lenticulus is made to have a diameter of 8.35 mm., a thickness of 2.4 mm. (that of the human lens is approximately 4 mm.) and the anterior and posterior radii of curvature are 17.8 and 10.7 mm. respectively whereas those of the human lens are 10 and 6 mm. A peripheral groove is cut into each surface of the lenticulus so that it may be grasped with a forceps. The lenticulus can be used after extracapsular and intracapsular extractions. If the extracapsular method is chosen great care is exercised to remove the anterior capsule of the lens completely with a toothed forceps and to wash out all cortical remnants so thoroughly that only the thin posterior capsule remains in the pupillary area. The corneoscleral wound is held open with an iris repositor and the acrylic lens which is held in a specially designed forceps is inserted behind the iris below. The lenticulus is then steadied with the iris repositor and the remaining portion of the iris is pulled over it by means of an iris hook. When the pupil is again round and central a peripheral iridectomy is done, the lenticulus is centered by pressure on the cornea and after final irrigation the two preplaced corneoscleral mattress sutures are tied.

Twenty-five eyes have undergone this operation. The first two were unsatisfactory because the lenticuli proved to be too strong optically. Of the others one was unsatisfactory because the corneoscleral wound in a feeble old man did not heal properly. Twenty-two eyes were surgically successful and binocular vision was better than after simple extraction alone. Several had fibrinous exudate with pigment but even in the worst eyes this is dispersing and disappearing completely. Now the vision is 6/36 in two eyes, one of which has myopic degeneration, 6/24 in one, 6/18 in three, 6/12 in two, 6/9 in five, 6/6 in five and 6/5 in two. In one case two years have elapsed since the lenticulus was introduced. Convalescence is not prolonged and the author has the impression that the dangers of this operation are comparable to those of a simple cataract extraction. A fuller account of this work will appear in the British Journal of Ophthalmology. F. H. Haessler.

11

RETINA AND VITREOUS

Fanta, H. **Vascular hypertension and the eye.** Wien klin. Wchnschr. 63:649-652, 1951.

This is a postgraduate lecture. The appearance of the fundus in hypertensive retinopathy caused by a dysfunction of the cerebral centers in cardio-cerebral disease is contrasted with the fundus in angiospastic retinopathy of renal and hematogenic origin. Hemorrhages are due to a damaged blood vessel wall rather than to the hypertension itself. Signs of arteriosclerosis, such as the arterio-venous crossing phenomenon may be present

without hypertension. Measuring the blood pressure in the central retinal artery may sometimes reveal an isolated regional hypertension. Two patients with severe attacks of headaches had normal blood pressure but very high pressure in the retinal artery. Treatment with "Hydergin (Sandoz)" seemed effective in 16 of 19 patients whose symptoms were similar to those of the patients described. (References) Max Hirschfelder.

Weinstein, Paul. **The significance of diabetic retinitis.** Ophthalmologica **121**: 353-356, June, 1951.

The systemic findings in 30 cases of diabetic retinopathy are reported. There was a high incidence of high capillary fragility, of abnormalities of the electrocardiogram and, in the cases of retinitis proliferans, of low creatinine clearance. Peter C. Kronfeld.

13

NEURO-OPHTHALMOLOGY

Bartlet, J. E. A. **A case of organized visual hallucinations in an old man with cataract, and their relation to the phenomena of the phantom limb.** Brain **74**: 363-373, 1951.

The author reviews the literature on hallucinations in patients with cataract and discusses the case of an old man with bilateral cataract who had well organized illusionary hallucinations distinguishable by him from reality. The syndrome of well-organized illusionary hallucinations is compared with the phantom limb phenomenon. An important factor in both syndromes is the absence of normal stimuli from the periphery. The deprivation of the part of the brain responsible for conscious impressions of its customary afferents may be an important factor in the production of these hallucinations. Theodore M. Shapira.

Hoff, H., and Seitelberger, F. **Clinical and pathological findings in bilateral involvement of the occipital lobes.** Wien. klin. Wchnschr. **63**:621-623, 1951.

A patient with vascular hypertension had a cerebral vascular accident with right hemiparesis, right hemianopsia, which spared the macula, alexia and impairment of the sense for numbers. The clinical diagnosis was thrombus of the left posterior cerebral artery. Eighteen months later a second hemorrhage caused an upper hemianopsia with macular involvement, optic agnosia, alexia, disturbance of optic attention and nonrealization of the optic disturbance. The clinical diagnosis of a fresh encephalomalacia in the region of the right posterior cerebral artery was confirmed by the findings at autopsy. (3 figures) Max Hirschfelder.

Psenner, L. **X-ray changes in opticochiasmatic arachnoiditis.** Wien. klin. Wchnschr. **63**:679-680, 1951.

Enlargement and irregularities of the optic canal may be found in the X-ray picture of certain cases of arachnoiditis. X-ray pictures of the entire skull may show sinusitis or fractures. The differential diagnosis between neoplastic and inflammatory intracranial disease is greatly aided by careful X-ray examination of the skull. (2 figures) Max Hirschfelder.

Walsh, F. B. **Ocular symptoms of migraine.** North Carolina M. J. **12**:271-273, July, 1951.

The author outlines the ocular symptoms of migraine and stresses the importance of understanding the patient's problem, gaining his cooperation and helping him to adjust his stresses and strains. Theodore M. Shapira.

Yourish, Norman. **Adie's syndrome.** U. S. Armed Forces M. J. **2**:1515-1517, Oct., 1951.

Adie's syndrome consists of a tonic pupillary reflex in which the pupil reacts very poorly to light and convergence, and the absence of knee and ankle reflexes in an otherwise healthy person. A 21-year-old man who had this syndrome with tonic pupil in the right eye is described. The pupil is not a truly fixed pupil but reacts very slowly and usually seems slightly dilated when compared with that of the other eye, although it may be smaller. The pupil reacts promptly and fully to all mydriatics and miotics. Routine tests of installation of 2.5 percent methacholine chloride resulted in prompt contraction of the tonic and no contraction of the normal pupil. The etiology is unknown and no treatment is needed.

Bennett W. Muir.

14

EYEBALL, ORBIT, SINUSES

King, A. B. **Traumatic encephaloceles of the orbit.** A.M.A. Arch. Ophth. **46**:59-56, July, 1951.

Traumatic encephaloceles of the orbit may occur through defects in the orbital roof produced by the intracranial approach for the removal of orbital tumors, or by trauma during which the floor of the anterior fossa is shattered and distorted. Infection of the protruding brain and of the subarachnoid space becomes a major hazard. A method of repair of the dura and bony defects is discussed and two cases are reported. (5 figures)

Edward J. Swets.

15

EYELIDS, LACRIMAL APPARATUS

Fazakas, S. **Modern principles underlying tear sac surgery.** Ophthalmologica **122**:10-17, July, 1951.

The author believes that the tear sac, but not the canaliculi, play an active part in the elimination of the tears. He bases his views on 1. roentgenologic studies by means of lipoidol, 2. manometric studies (connecting a lacrimal cannula with a water manometer) and 3. the direct observation of excursions of the lateral tear-sac wall during lid movements. During lid closure the lacrimal portion of the orbicularis pulls the lateral wall of the sac laterad and thereby opens and distends the sac; when the lids open, Horner's muscle relaxes, permitting the lateral wall of the sac to relax also and to come in contact with the fixed medial wall. No surgery should be undertaken on the tear-sac without accurate knowledge of the function of the sac. If the manometer test shows no pressure changes and the lipoidol shadow no changes in size as the result of lid movements, the operation aiming at the restitution of the tear drainage will no doubt fail. The only useful procedure is the removal of the sac. If, on the other hand, definite pressure and roentgenologic shadow variations occur with lid movements, restitution of normal patency of the nasolacrimal duct should be attempted by gradual dilation; if this fails dacryocystorhinostomy is indicated.

The usual landmarks in tearsac surgery, that is the internal canthal ligament, the anterior lacrimal crista and the lacrimal fossa, are not as reliable as is generally believed. In 21 percent of over 1,000 skulls examined anomalies existed with regard to the position of these landmarks which could have led the surgeon astray. The author recommends the upper aperture of the bony nasolacrimal canal as an infallible landmark: to facilitate the union between the mucosa of the sac and that of the nose he mobilizes and places the sac into the rhinostomy.

Peter C. Kronfeld.

NEWS ITEMS

Edited by DONALD J. LYLE, M.D.
601 Union Trust Building, Cincinnati 2

News items should reach the editor by the 12th of the month but, to receive adequate publicity, notices of postgraduate courses, meetings, and so forth should be received at least three months before the date of occurrence.

DEATHS

Dr. Joseph V. Clothier, Pocatello, Idaho, died October 16, 1951, aged 72 years.

Dr. Norman Darrell Harvey, Pelham, New York, died October 13, 1951, aged 86 years.

Dr. Isaac David Kruskal, Brooklyn, New York, died recently, aged 71 years.

Dr. Walter Davis Stevenson, Quincy, Illinois, died October 26, 1951, aged 67 years.

ANNOUNCEMENTS

SILVER ANNIVERSARY CONGRESS

At the 25th annual spring congress in ophthalmology and otolaryngology of the Gill Memorial Eye, Ear, and Throat Hospital, Roanoke, Virginia, April 7 to 12, a number of outstanding participants will be on the program. The guests of honor are Dr. T. Keith Lyle of London, England; Dr. E. C. Kendall, discoverer of cortisone; Surgeon General Lamont Pugh, Dr. Edwin B. Dunphy, Dr. John H. Dunnington, Dr. Bayard T. Horton, Dr. Francis H. Adler, Dr. Henry M. Goodyear, Dr. Chevalier L. Jackson, Dr. Howard P. House, and Dr. Frederick A. Figi.

MISCELLANEOUS

INTERNATIONAL TRACHOMA PRIZE

The International League against Trachoma will give an international prize of 100,000 French francs in 1952 for the best work on etiology, pathogenesis, pathologic anatomy, or physiology of the trachomatous eye. The award was created by Mrs. Cuenod and Roger Nataf of Tunis. Five copies of the work accompanied by an abstract of four typewritten pages in French, English, Italian, and Spanish, must be sent to the Secretary General, Dr. Jean Sedan, 94, Rue Sylvabelle, Marseilles, before March 1, 1952. The winning work will be published in various languages in the *Revue Internationale du Trachome*. The prize will be awarded at the general meeting of the Congress of the Société Française d'Ophthalmologie in 1952.

NSPB GRANTS FOR RESEARCH

The National Society for the Prevention of Blindness has made grants totaling $8,942 for research on eye diseases and defects that can lead to blindness. At present only a few hundred thousand dollars is spent annually in the United States for research on blindness. In contrast, the American public last year spent 125 million dollars for pensions and care of those already blind.

Grants went to the Massachusetts Eye and Ear Infirmary, Boston; Western Reserve University, Cleveland; and Washington University School of Medicine and McMillan Eye Clinic, St. Louis. A portion of the funds in the Western Reserve and Washington University grants came from a New Jersey organization, New Eyes for the Needy, which is coöperating in the national research program.

NATIONAL COUNCIL TO COMBAT BLINDNESS GRANTS-IN-AID

The National Council to Combat Blindness, in accordance with its program of supporting ophthalmic research, is now receiving applications for its third annual grant-in-aid awards, to be made in June, 1952. Applications to be considered for this group of awards should be in the office of the council no later than April 15, 1952. Application forms may be obtained by addressing: Secretary, National Council to Combat Blindness, 1186 Broadway, New York 1, New York.

The organization's committee on research, consisting of: Chairman, Dr. Phillips Thygeson, University of California, San Francisco; Dr. Alson E. Braley, State University of Iowa College of Medicine, Iowa City; Dr. Dan M. Gordon, Cornell University Medical College, New York; Dr. Stuart Mudd, University of Pennsylvania School of Medicine, Philadelphia; Dr. Charles A. Perera, Columbia University College of Physicians and Surgeons, New York; and Dr. Samuel L. Saltzman, New York Medical College, Flower and Fifth Avenue Hospitals, awarded the following grants-in-aid in 1951:

Retina Foundation, Massachusetts Eye and Ear Infirmary, Boston, $5,000, Dr. Endre A. Balazs, acid mucopolysaccharides in the vitreous body, their role in the composition of the vitreous body, and their relationship to the proteins in the vitreous gel; University Hospitals, University of Iowa, Iowa City, $2,700, Dr. Hermann M. Burian, study of the electrical response of retina and brain in patients with amblyopia ex anopsia and suppression; New York University Post-Graduate Medical School, New York, $1,296, Dr. Helena Fedukowicz, attempts to grow the virus of inclusion conjunctivitis, follicular conjunctivitis and folliculosis; New York Eye and Ear Infirmary, New York, $1,600, Dr. Louis J. Girard, focal infection clinic; State University of Iowa Medical School, Iowa City, $1,200, Dr. Ralph C. Janes, a study of the incidence and basic cause of cataract, hemorrhages, and degenerative changes in the retina and optic nerve of alloxan diabetic animals; University of California Medical School, $1,200, Dr. Samuel J. Kimura, the relationship of uveitis to bacterial allergy; University of Pennsylvania, Philadelphia, $2,500, Dr. H. P. Kirber, virus infections of ocular

tissues grafted onto the chorioallantoic membrane of the chick embryo; Government Hospital, Haifa, Israel, $2,500, Dr. I. C. Michaelson, factors affecting new vessel growth into the cornea; Indiana University Medical School, Indianapolis, $1,500, Dr. T. F. Schlaegel, Jr., studies on the mechanism of development of contralateral granulomatous uveitis from the use of horse serum in rabbits; New York Hospital, Cornell Medical Center, New York, $750, Dr. Edward Tolstoi, retinopathy in diabetes; New York Medical College, Flower and Fifth Avenue Hospitals, New York, $466, Dr. Charles Haig, continuance of study on regional light sensitivity of the retina.

Dr. James H. Allen of Tulane University, New Orleans, has joined the organization's Committee on Research.

Yale postgraduate meeting

Dr. Frederick H. Theodore, assistant clinical professor of ophthalmology, Post-Graduate Medical School, New York University, Bellevue Medical Center; assistant ophthalmic surgeon, Manhattan Eye, Ear, and Throat Hospital; adjunct ophthalmic surgeon, Mount Sinai Hospital, was guest lecturer on December 15, 1951, at the Yale University School of Medicine Post-Graduate Courses in Ophthalmology. His subject was "Allergic conjunctivitis." The lecture was followed by Kodachrome pictures illustrating the different types of allergic conjunctivitis and a brief discussion of the treatment was given.

The meeting was preceded by discussion of two clinical cases:

1. A 13-year-old white boy with huge cobblestone palpebral lesions with an unconfirmed diagnosis of vernal conjunctivitis. Scrapings have been negative for eosinophils; there was little itching; response to all medication including local cortisone had been poor. Systemic cortisone will be tried.

2. A 57-year-old man, a known diabetic and borderline hypertensive, with a five-week febrile course unyielding to all antibiotic treatment, had a blind, nonpainful eye, with chronic uveitis, detachment, exophthalmos, and poor visualization of the fundus. He was presented because of questionable nodules in the liver. The question of a malignant melanoria or liver abscess was the serious differential diagnosis. Transillumination was not helpful.

Research Study Club program

The 21st annual midwinter postgraduate clinical convention of the Los Angeles Research Study Club was held January 13 through 25 at Los Angeles. Beginning Monday, January 21, the following eye program was presented:

Sir Stewart Duke-Elder: "The problem of glaucoma," "The natural history of simple glaucoma," "The natural history of congestive (narrow-angle) glaucoma," "The secondary glaucomas," "The diagnosis of glaucoma," "The medical treatment of glaucoma," and "The surgical treatment of glaucoma."

Dr. Isaac H. Jones, "Antibiotics: Two new cautions"; Dr. Clarence H. Albaugh, "Practical considerations of geriatrics and metabolic influences on the eye" and "Recent developments in the use of cortisone in ophthalmology"; Dr. John A. Bullis, "The mechanical effect of extraocular muscle surgery"; Dr. William J. Endres, "Selection of surgical procedures in glaucoma"; Dr. A. Ray Irvine, Jr., "Mistakes commonly made in clinical ophthalmology as revealed by pathologic examination"; Dr. Robert J. Schillinger, "Calibration of the trial case to eliminate induced hyperphoria"; Dr. Carrol L. Weeks, "Practical aspects of retinal separation surgery."

Dr. Warren A. Wilson, "Retrolental fibroplasia: Recent concepts" and "Evaluation of office orthoptic procedures"; Dr. Harold F. Whalman, "Developmental anomalies of the human eye"; Dr. S. Rodman Irvine, "Recent medico-ophthalmic concepts of arteriosclerosis, diabetes, hypothyroidism, and myasthenia gravis" and "Late vitreous changes following cataract extraction."

Dr. Etta C. Jeancon, "The problem of eye accidents in small children"; Dr. Harold G. Scheie, "Gonioscopy," "The management of congenital glaucoma," and "Cyclodiathermy"; Dr. Joseph G. Smith, "Successful treatment of ocular methylrosaniline (indelible pencil) poisoning by means of fluorescein solution" and "A method of avoiding hammock pupil in cases of vitreous loss in cataract extraction."

Dr. M. N. Beigelman, "Pseudotumors of the macular region"; Dr. George P. Landegger, "Complications following cataract surgery: Parts I and II"; and Dr. Joseph H. Failing, "General anesthesia in cataract surgery." Dr. Meyer Wiener gave a special course in surgery of the eye.

Societies

Madrid officers

Officers of the Madrid Ophthalmological Society for the coming year are: President, Dr. Manuel Marin-Amat; vice-president, Dr. Jacinto Valentin Gamazo; director, Dr. Rafael Bartolozzi Sanchez; treasurer, Dr. Manuel Rios Sasiain; secretary, Dr. Fermin Galindez Inglesias.

Philadelphia officers

The Section on Ophthalmology of the College of Physicians of Philadelphia announces the election of the following officers: Chairman, Dr. George F. J. Kelly, 1930 Chestnut Street, Philadelphia 3; clerk, Dr. M. Luther Kauffman, Medical Arts Building, Jenkintown, Pennsylvania.

Brooklyn program

At the 117th regular meeting of the Brooklyn Ophthalmological Society, Dr. Arthur Gerard DeVoe presented a paper on "The problem of epicanthus," and Dr. John N. Evans spoke on "Use of refraction curve in daily practice."

Officers of the society are: President, Dr. Daniel K. Kravitz; vice-president, Dr. Donald E. Swift; secretary-treasurer, Dr. Louis Freimark; associate secretary-treasurer, Dr. George A. Graham.

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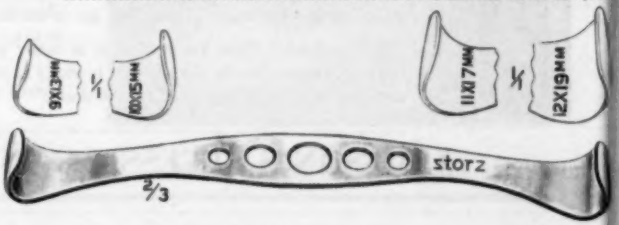

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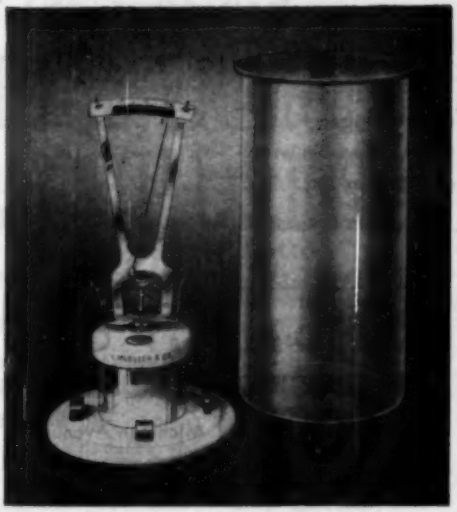

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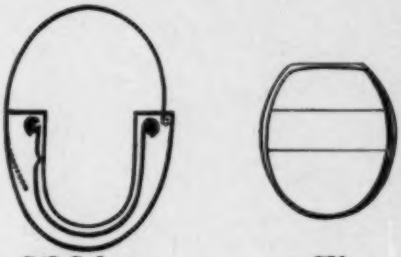

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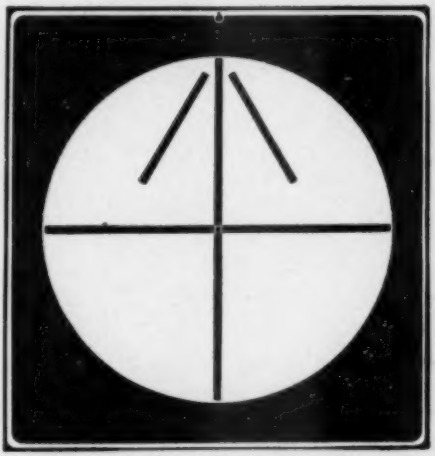

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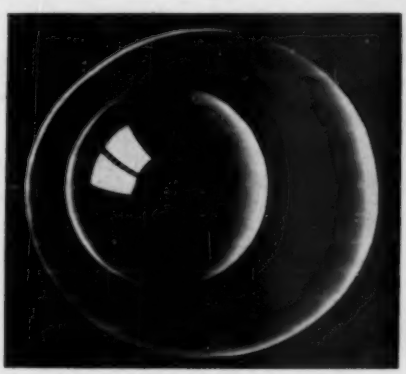

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